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Differential Diagnoses Of Temporal Bone Defects And Zygomatic Bone Lesions Found In Fetal And Infant Individuals From The Kellis 2 Cemetery, Dakhleh Oasis, Egypt

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DIFFERENTIAL DIAGNOSES OF TEMPORAL BONE DEFECTS AND ZYGOMATIC BONE LESIONS FOUND IN FETAL AND INFANT INDIVIDUALS FROM THE KELLIS 2 CEMETERY, DAKHLEH OASIS, EGYPT

by

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ABSTRACT

The Kellis 2 cemetery site within the Dakhleh Oasis, Egypt provides a unique study opportunity due to the large number of infant, perinatal, and fetal individuals that have been recovered. Several of the infant and fetal remains have undiagnosed circular defects on the temporal bone, and others have undiagnosed lesions on the zygomatic bone. Of the 268 individuals under one year of age that have been analyzed from the Kellis 2 cemetery, twenty-six individuals have the temporal bone defect and six have the zygomatic bone lesions. A survey of clinical and paleopathological research provided possible pathological conditions that could cause abnormalities such as defects or lesions on the temporal bones or zygomatic bones in the fetal and infant population. For this study, the temporal bone defects and zygomatic bone lesions were macroscopically observed and a descriptive analysis was created. The information garnered from the literature survey was then compared to the individuals from the Kellis 2 cemetery that had the temporal bone defects and zygomatic bone lesions to create a differential diagnosis. A differential diagnosis of the temporal bone defects includes mastoid emissary vein defects and petrosquamous sinus anomalies. A differential diagnosis of the zygomatic bone lesions includes scurvy. Contributing factors may also have been present in order for these defects and lesions to occur. Creating a differential diagnosis of the defects and lesions can provide information on the health, growth, and morbidity of the youngest members of the society related to the Kellis 2 cemetery.

In Loving Memory of Elizabeth Jardine and John Decknick

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CHAPTER ONE: INTRODUCTION

Children are often underrepresented in the archaeological record (Lewis, 2007). Studying the skeletal remains of children provides information regarding health, diet, growth and development, age at death, and social and economic information that can contribute to interpretations surrounding trauma and disease (Lewis, 2007). Juvenile physical remains provide the most direct connection to their past lives (Lewis, 2007). Fetal skeletons are considered a rarity in archaeology, most likely due to poor recovery techniques, misidentification, and poor preservation (Tocheri et al., 2005). Cultural bias and exclusion also result in fewer non-adult skeletal remains being recovered (Lewis, 2007). The Kellis 2 cemetery site within the Dakhleh Oasis, Egypt provides a unique study opportunity due to its large collection of individuals less than one year old (Tocheri et al., 2005). A portion of these skeletons are infant, perinatal, and fetal skeletons that have undiagnosed circular defects on the temporal bone, and others that have undiagnosed lesions on the zygomatic bone. Interpretation of data in human skeletal paleopathology depends on the accuracy of the differential diagnosis of the diseases apparent in the archaeological skeletal remains (Ortner, 2003). Creating differential diagnosis of these defects and lesions can provide information on the health, growth, and morbidity of the youngest members of the society related to the Kellis 2 cemetery.

The Kellis 2 cemetery is part of the excavation and research focus of the Dakhleh Oasis Project, an ongoing project which involves the excavation and analyses of many sites in the Dakhleh Oasis, Egypt (Bagnall, 2001; Cook, 1994; Figure 1). The Dakhleh Oasis Project has conducted field seasons since 1978, involving multi-disciplinary investigations (Cook, 1994). The bioarchaeology team of the Dakhleh Oasis Project has partially excavated the two cemeteries associated with the ancient village of Kellis, and in particular, systematic

excavations at the Kellis 2 cemetery began in 1992 (Birrell, 1999). These two cemeteries are associated with ancient Kellis, referred to as the West and East Cemeteries or as Kellis 1 and 2 (Birrell 1999). The cemeteries are east and west of a wadi; the East cemetery (K2) on a broad plain and the West Cemetery (K1) on a progression of low hills (Birrell, 1999). The climate of the Dakhleh Oasis is considered to have been hyperarid during the periods of occupation related to the cemeteries (Molto, 2002). During the Roman period of occupation, the Dakhleh Oasis' population was larger than in previous periods (Molto, 2002). The population at the town site of Kellis probably reached several thousand during the 4th century AD (Hope, 2001). Despite the increase in population, villages like Kellis were still considered to be small, not overcrowded, and widely spaced (Molto, 2002). Based on the archaeological evidence of the different mortuary practices found at the two cemeteries, Kellis 2 is considered to be an early Christian cemetery (Bowen, 2003).

The East Cemetery, or Kellis 2, is located on a plateau that is three meters higher than the main wadi (Birrell, 1999). The approximate dimensions of the Kellis 2 cemetery have been determined to be about 150 m in the east-west direction, and 60 m in the north-south direction, although this is currently still under investigation (Birrell, 1999). Kellis 2 consists of interspersed tomb structures, with singular rectangular pit graves that were cut into the red Nubian clay (Birrell, 1999). Tentative radiocarbon dating of the excavated pit graves date the Kellis 2 cemetery to the late third to early fourth centuries CE, during the Roman-Byzantine period (Birrell, 1999; Molto, 2001). Though heavily debated, the radiocarbon dates for the Kellis 2 site indicates that perhaps the cemetery was in use for longer than first indicated despite the lack of archaeological evidence to support the radiocarbon dates (Stewart et al., 2003).

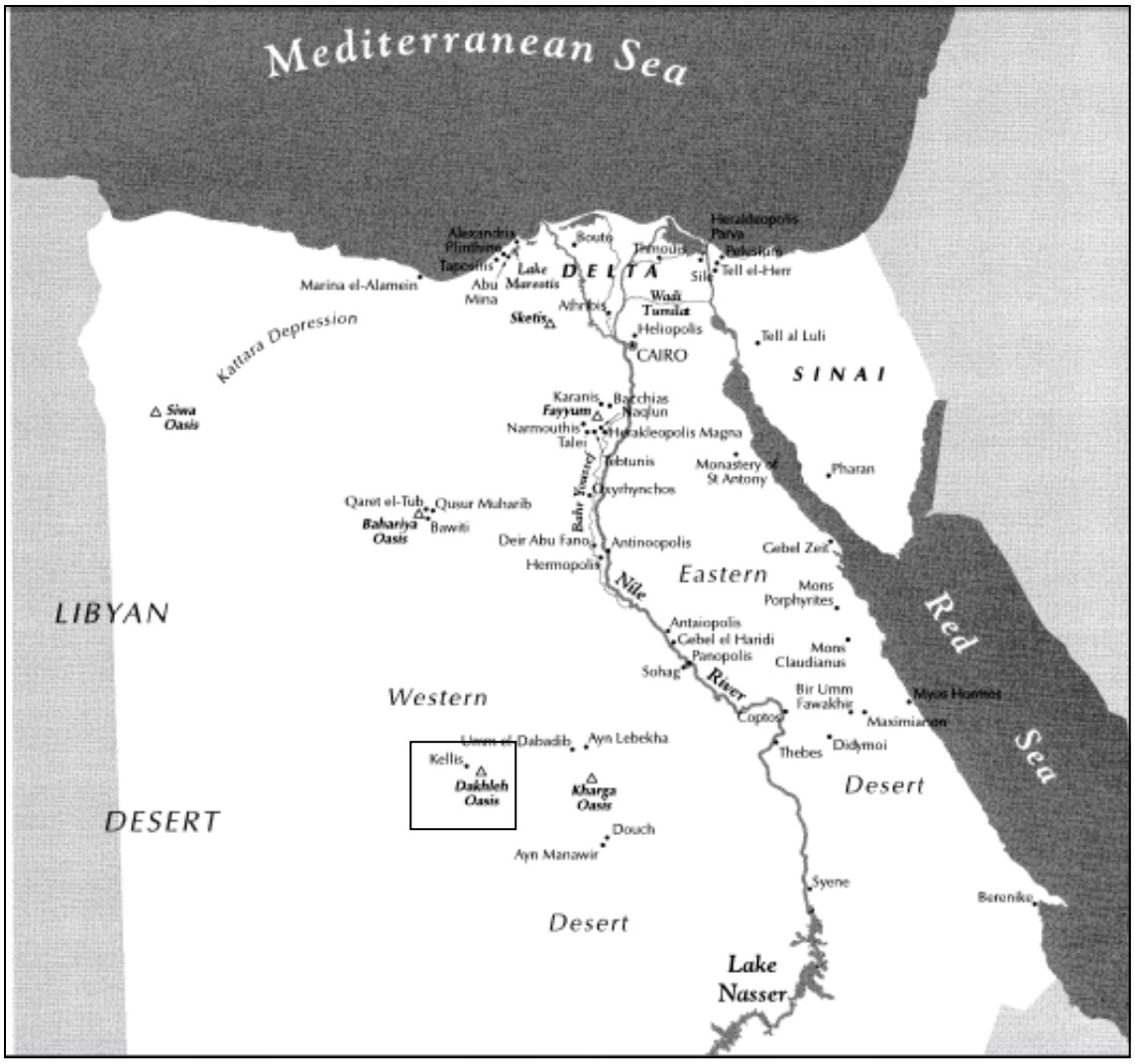


Figure 1: Map of Egypt with the location of the Dakhleh Oasis (adapted from Bagnall, 2001)

In almost all cases the graves have single inhumations, with the head orientation to the west and the bodies placed in an extended, supine position (Birrell, 1999). A few graves had ceramic coffins and one had a reused wood coffin; however, coffins were not generally used in the Kellis 2 cemetery (Wheeler, 2009). In many cases the bodies were reduced to skeletons with their linen wrappings; however the linen wrappings around infant bodies survived in better condition (Birrell, 1999). In some instances the environmental conditions and burial practices resulted in the preservation of soft tissues such as skin, muscle tissue, internal organs, hair and

nails (Williams, 2008). Children were buried in shallower burial pits than the adults, and therefore many were exposed to wind erosion (Birrell, 1999). There is evidence that roughly half of the burials that have been excavated were disturbed by looting (Wheeler, 2009).

As of 2009, 701 individuals have been recovered from the Kellis 2 cemetery, and the demographic profile suggest that 65% of the population is juvenile and 35% is adult (Wheeler, 2009). Infants were occasionally found with some broken pottery placed over or near the face, or in some cases the broken pottery covered the entire grave (Birrell, 1999). The fetal, perinatal, and neonatal remains are distributed throughout the cemetery (Figure 2; Tocheri et al., 2005). These remains are buried amongst and in a similar manner as the adults and other juveniles (Tocheri et al., 2005). These characteristics indicate that the mortuary practices of the ancient inhabitants of Kellis included the burials of individuals of all ages, and therefore resulted in similar mortuary practices for all (Bowen, 2003; Marlow, 2001).

Kellis 2 is believed to be an early Christian cemetery due to the dating of the site, orientation of the graves, few or absent grave goods, inclusion of all ages of individuals, the presence of burial shrouds, and single internments (Bowen, 2003; Bowen, 2004). Because of the mortuary treatment, the Kellis 2 cemetery provides a unique opportunity to study fetal skeletal remains. The large number of fetal remains at Kellis 2 is attributed to a combination of the arid environment and the cultural treatment of the fetal, neonatal, and perinatal remains during this time period (Tocheri et al., 2005). Of the juveniles recovered from the Kellis 2 cemetery, 104 were estimated to be between fetal and birth ages, and an additional 164 were estimated to be between birth and twelve months of age (Wheeler, 2009). During the initial examination of the juvenile remains, it was noted that several individuals had circular defects on their temporal bones, and others had lesions on their zygomatic bones (Wheeler, pers. comm.).

Both of these conditions have not been documented in the literature, and thus are the focus of this thesis.

The primary goal of this thesis is to present a differential diagnosis for both the circular defects found on the temporal bones and the lesions on the zygomatic bones of fetuses, perinates, and infants from the Kellis 2 cemetery at the Dakhleh Oasis, Egypt. These two conditions that are found in the individuals from the Kellis 2 cemetery appear to be unrelated. The complicated relationship between growth and development and age (Scheuer and Black, 2000) may also be related to the cause of the circular defect of the temporal bone and the cause of the lesion on the zygomatic bone. In Chapter Two, the individuals are identified and the method of examination is determined. In Chapter Three the developmental process of the temporal and zygomatic bones of the fetus and infant will be examined for possible correlations and/or causes of the undiagnosed defect and lesion. In Chapter Four, medical and paleopathological literature are reviewed for determining a differential diagnosis. In Chapter Five, the results of the macroscopic observation are analyzed. A differential diagnosis for the temporal bone defect and the zygomatic bone lesion will then be established in Chapter Six. In the final and seventh chapter, the differential diagnoses will be summarized and future considerations will be discussed. Previous analysis of the Kellis 2 population determined that there was a low life expectancy at birth, most likely due to the harsh living conditions (Molto, 2001). By establishing a differential diagnosis of the unidentified circular defects on the temporal bones and the unidentified lesions on the zygomatic bones of fetuses and perinates, a clearer picture can be developed on the living conditions of the Kellis settlement and how it affected endogenous and exogenous health.

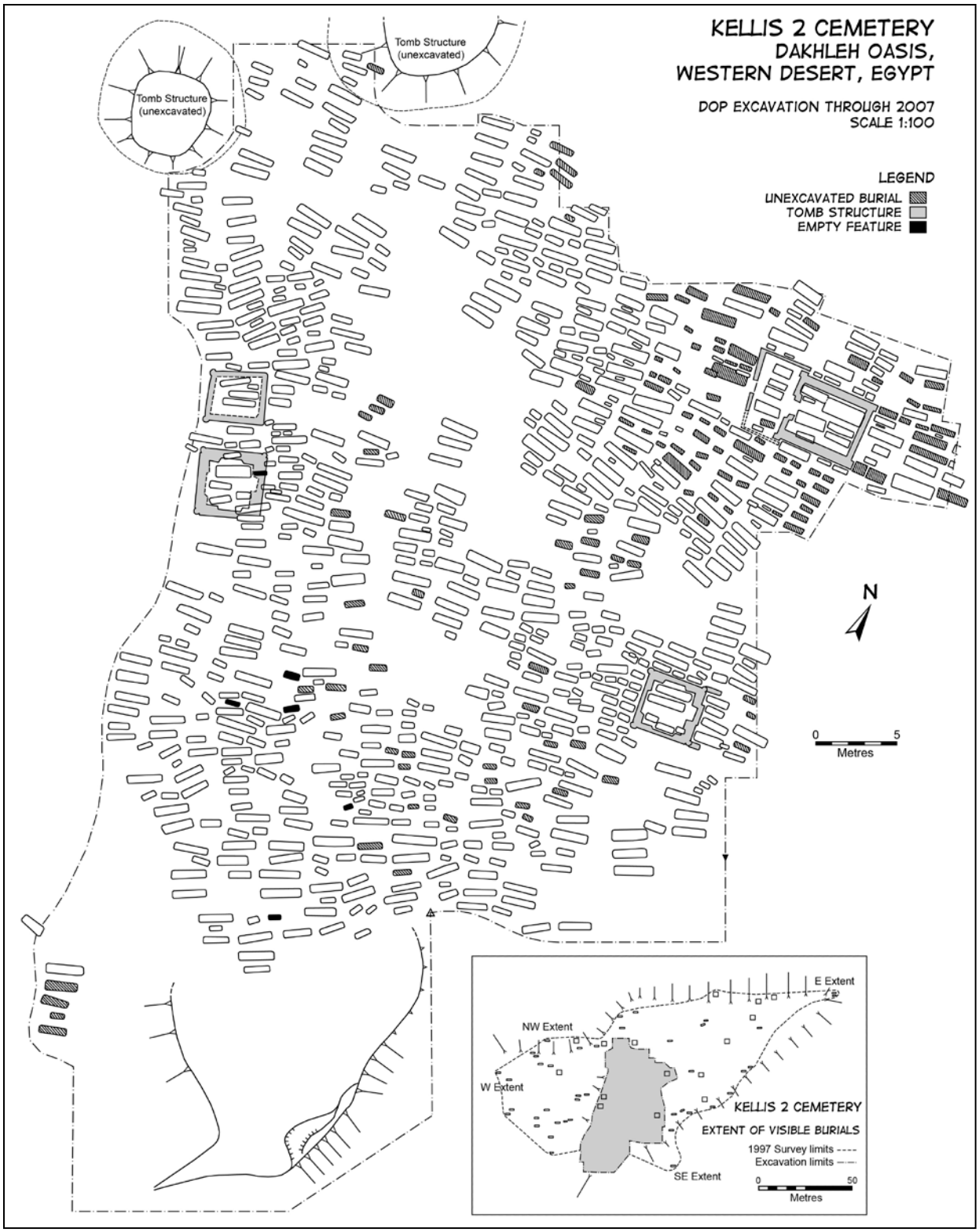


Figure 2: Kellis 2 cemetery of the Dakhleh Oasis, Egypt (Map courtesy of L. Williams)

CHAPTER TWO: MATERIALS AND METHODS

The age classifications of this sample must be clarified to avoid the variability that occurs in age terminology both within and among the disciplines. The age definitions used for this thesis are based upon pediatric age (Table 1; Scheuer and Black, 2000; Lewis, 2007). A fetus is generally described as from 8 weeks of gestational or intrauterine life until birth (Scheuer and Black, 2000). Modern medicine considers the birth of a fetus prior to 38 weeks gestational age as premature, due to the health complications and decreased chance of survival that occurs in births prior to 38 weeks gestational age. Thus, full term is established as 38-42 weeks. Determining the age of such skeletal remains is important, for an estimated age could indicate that the skeletal remains were viable at birth or premature.

Table 1: Age terminology (after Scheuer and Black, 2000; Lewis, 2007)

Term	Description
Embryo	First 8 weeks of intra-uterine life or gestation
Fetus, fetal	From 8 weeks gestation until birth
Perinatal, perinate	Around the time of birth, from 24 weeks gestation to 7 days after birth
Neonatal, neonate	From birth to 28 days
Premature	Before 38 weeks gestation
Full term	From 38 to 42 weeks gestation
Infant	From birth to one year of age
Juveniles	Younger than about 15 years of age
Adults	Older than about 15 years of age

Terminology used in reference to abnormal bone conditions can differ between clinical and paleopathological literature, so the terminology used in this paper regarding the abnormal appearances of the zygomatic and temporal bones must be more clearly defined. In this paper, ‘abnormal bone’ or ‘bone abnormalities’ refers to the presence of a lesion, defect, or other anomaly that represents a variation from the normal anatomical appearance of the bone. These

variations provide the initial evidence of a possible pathological condition (Ortner, 2003). Lesion is often used to refer generally to abnormal bone (Ortner, 2003). Lesion can also be used in a more limited manner, defined as the tissue manifestations of a specific disease (Roberts and Manchester, 2005). Lesions can be divided into categories: lytic, proliferative, and deformative (Ortner and Putschar, 1981). A defect is often a result of a disruption of the normal growth process of bone from environmental or congenital factors (Ortner and Putschar, 1981); an opening is present the inner or outer (or both) tables of the bone as a result of the disruption in the normal growth (Ortner and Putschar, 1981).

Kellis 2: Bioarchaeological Context

The individuals examined in this study are from the skeletal population of the Kellis 2 cemetery, Dakhleh Oasis, Egypt. The Dakhleh Oasis Project began its multidisciplinary and systematic investigation of the Kellis 2 cemetery in 1992 (Birrell, 1999). The ancient town of Kellis, believed to have buried its dead in the Kellis 2 cemetery, reached its peak population of several thousand during the fourth century AD (Hope, 2001). During the Roman period, juveniles typically made up a large percentage of cemetery populations (Wheeler, 2009). The Kellis 2 cemetery follows that trend with approximately 65% of 701 excavated individuals having been classified as juveniles (Wheeler, 2009). The Kellis 2 cemetery is one of few archaeological contexts that provide the ability to study fetal remains at the population level (Tocheri et al., 2005). Each gestational age category is represented in the fetal population found in the Kellis 2 cemetery, which suggests that stillbirths and neonatal deaths received the same burial treatment (Tocheri et al., 2005). Also, the graves of the juvenile population appear to be equally distributed throughout the Kellis 2 cemetery (Wheeler, 2009; Figure 3). Analysis of the burials from the Kellis 2 cemetery shows that the individuals were placed directly onto the floor of the grave with the head to the west (Bowen, 2003). Infant burials were found in shallow graves, and

the practice of placing the individuals in an east-west orientation was also continued in the infant burials (Bowen, 2003). This practice of an east-west orientation is an early Christian tradition, and the Kellis 2 cemetery is therefore identified with the early Christian community associated with ancient Kellis (Bowen, 2003).

The juvenile population represents over half of the total population that has been excavated at the Kellis 2 cemetery, and almost half of the juvenile population is represented by the infant and fetal age categories (Wheeler, 2009; Figure 4). Much research has been conducted on the juvenile population from the Kellis 2 cemetery, including bioarchaeological analyses, weaning practices, isotopic analyses, and seasonality of mortality (Wheeler, 2009; Wheeler, 2010; Williams, 2008; Dupras et al., 2001; Dupras and Tocheri, 2007). The juvenile population from the Kellis 2 cemetery has been studied for signs of physiological stress such as cribra orbitalia, dental enamel hypoplasia, and osteoperiostitis (Wheeler, 2010). Though these are non-specific stress indicators, they can still aid in identifying the general health of the juvenile population during the political, ideological, economic, and dietary shifts during the Romano-Byzantine period in Egypt (Wheeler, 2010). The determination of the seasonality of juvenile mortality at the Kellis 2 cemetery based on grave orientation has shown that there was a spring seasonal mortality peak (Wheeler, 2009). Based upon stable isotope analysis of naturally mummified tissues and the solar alignment of graves, infants were found to have had a higher seasonal incidence of mortality in warmer seasons in the Dakhleh Oasis (Williams, 2008).

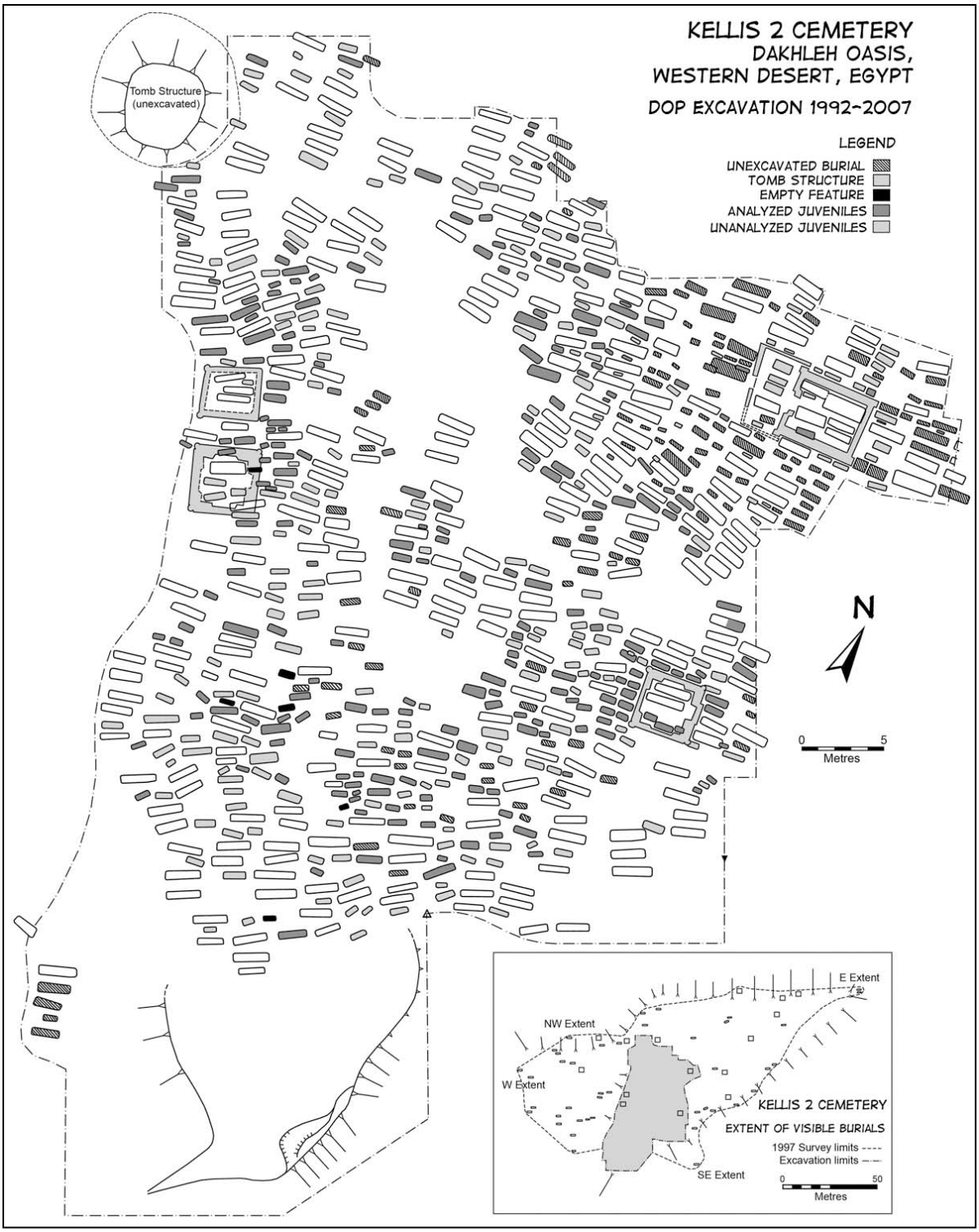


Figure 3: The distribution of juveniles in the Kellis 2 cemetery, Dakhleh Oasis, Egypt. (Williams, 2008; Wheeler, 2009)

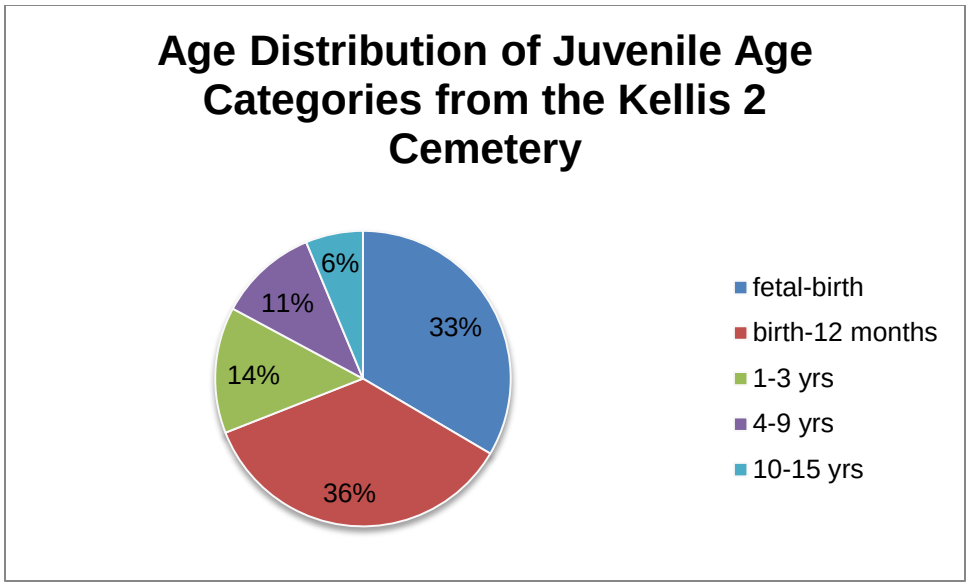


Figure 4: Age distribution of the juvenile individuals from the Kellis 2 cemetery (data adapted from Wheeler, 2009)

Materials

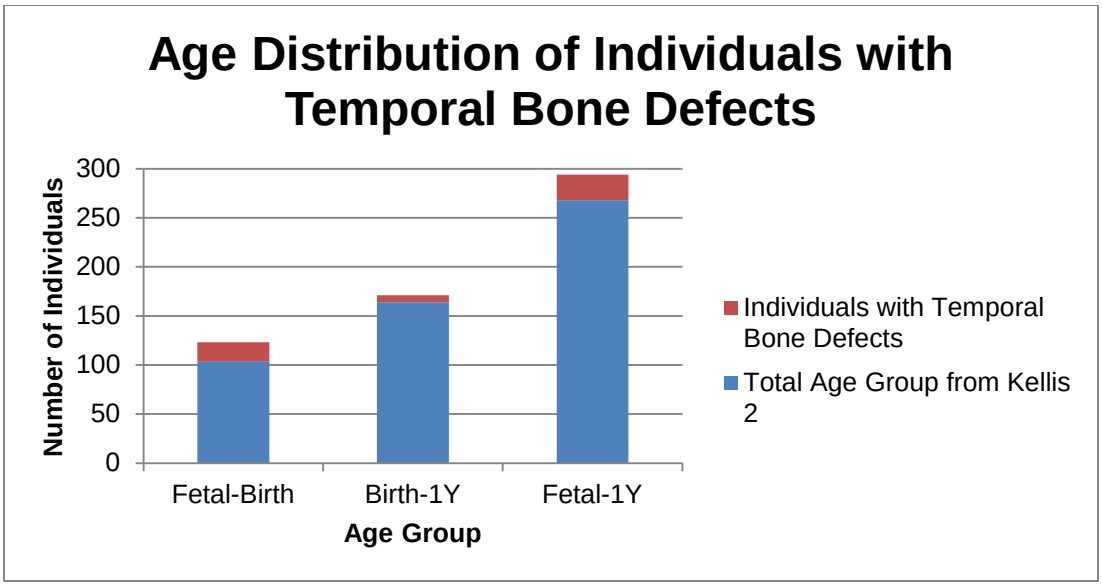
Of the juvenile population recovered from the Kellis 2 cemetery, 104 individuals are estimated to have been between fetal to birth ages at death (Wheeler, 2009). An additional 164 are estimated to have been between birth and 12 months old at death (Wheeler, 2009). Therefore, there are roughly 268 infant and fetal individuals that have been recovered from the Kellis 2 cemetery (Wheeler, 2009). Of these, thirty-two individuals were found to have the temporal and zygomatic bone abnormalities. Twenty-six individuals (9.7%) from the infant and fetal population of the Kellis 2 cemetery have the circular defect on their temporal bones (Table 2, Figures 5 and 6). Six individuals (2.2%) from the infant and fetal population from the Kellis 2 cemetery have the unidentified lesions on their zygomatic bones (Table 3, Figures 7 and 8). These individuals were identified as having the undiagnosed temporal bone defects and zygomatic bone lesions based upon the location and appearance of the abnormalities, and the

lack of a diagnostic explanation (Wheeler, pers. comm.). The estimated age at death for these individuals were previously determined by Wheeler (2009).

Table 2: The twenty-six fetal and neonatal individuals, sorted by age, from the Kellis 2 cemetery that were found to have the temporal bone defect. (Age based on age range estimations; adapted from Wheeler, 2009)

Burial Number	Age	Age Range
671	32w	30.4-42.4w
318B	34w	33.6-34.5w
575B	34w	34.3-35.1w
701	35w	33.4-35.6w
313	37w	35.9-37.1w
154	37w	36-38w
436	37w	36.4-36.9w
29	38w	36.1-41.5w
387	38w	38.2-38.4w
462	38w	36.9-38.9w
513B	38w	38.1-39.2w
518	38w	37.2-41.0w
537	38w	37.1-39.9w
572	38w	37.6-38.4w
17	39w	36.7-41.3w
142	39w	37.2-42.4w
495	39w	37.0-43.1w
7	40w	36.4-44.2w
151	40w	39.0-43.2w
419	40w	39.4-41.2w
504	1m	birth-2m
660	1m	birth-2m
420	1m	birth-2m
508	1m	birth-2m
118	1m	birth-2.4m
123B	2m	1-3m

(w = weeks gestation; m = months of infancy)



(Y= years of age)

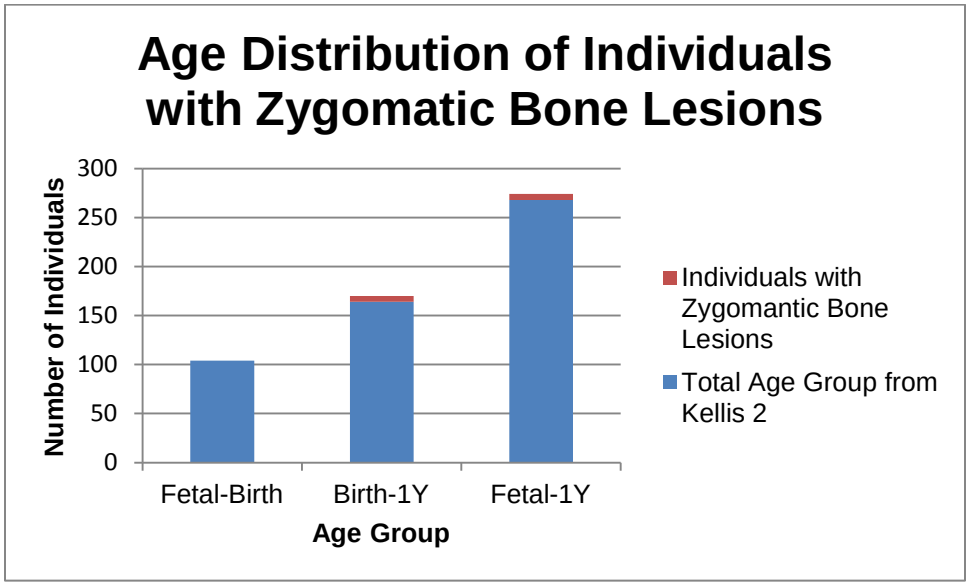
Figure 5: The distribution of the individuals with the temporal bone defect from the Kellis 2 cemetery and their estimated ages at death.

Figure 6: Left and right temporal bones from individual 318 from the Kellis 2 cemetery from the Dakhleh Oasis, Egypt; a typical example of the temporal bone defects (Image courtesy of S. Wheeler).

Table 3: The six neonatal and infant individuals from the Kellis 2 cemetery, sorted by age, which were found to have the zygomatic bone lesion (Age is based on age range estimations; adapted from Wheeler, 2009)

Burial Number	Age	Age Range
577	40w	perinatal
580	1m	Birth-2m
118	1m	Birth-2.4m
575A	2m	1-3m
390	6m	6m + 3m
574	6m	.4 - .5Y

(w = weeks gestation; m = months of infancy; Y = years of age)



(Y= years of age)

Figure 7: The distribution of the individuals with the zygomatic bone lesion from the Kellis 2 cemetery based upon their estimated ages at death.

Figure 8: Left and right zygomatic bones from individual 580 from the Kellis 2 cemetery, Dakhleh Oasis, Egypt; a typical example of the zygomatic bone lesions (image courtesy of Wheeler)

Methods

All skeletal material from the Kellis 2 cemetery was examined using macroscopic observation. This method allows a non-destructive way to examine the skeletal material and therefore allows the skeletal material to be preserved for future studies. By observing the gross appearance of the temporal bone defects and zygomatic bone lesions, a descriptive analysis can be undertaken. The first step in macroscopic observation is to determine whether or not the abnormalities are postmortem or antemortem. The bone abnormality occurs should then be classified as woven, compact, or spongy (Ortner, 2003). The border around the bone destruction needs to be observed for characteristics such as no margin or a clear margin, no repair or evidence present of repair (sclerosis and no sclerosis), central destruction with marginal formation, repair in the central destruction, focal or general porous destruction, destructive remodeling, presence of osteopenia or osteoporosis, and if there are fractures or not (Ortner, 2003). These characteristics are important components in identifying the underlying

etiology of the abnormality. The goal of any descriptive analysis should be to identify the disease process, but even if the etiology of the bone abnormality cannot be specifically identified then the description of the abnormality is still important in documenting its paleopathological appearance and frequency (Ortner, 2003).

After the macroscopic examination takes place, then a differential diagnosis can be conducted based upon the characteristics of the abnormality. The age of the individuals that were found to have the abnormalities is also important in establishing a differential diagnosis. A literature survey was performed to determine what possible conditions could or could not be responsible for the observed abnormalities. From both clinical and paleopathological sources, conditions were noted that occur in the temporal or zygomatic bones. The pathological conditions that were found to occur in the temporal and/or zygomatic bones include infections, systemic diseases, metabolic diseases, bone diseases, neoplasms, and trauma. Considering the age range of the individuals who have the temporal and/or zygomatic bone abnormalities, congenital conditions were also included. The pathological conditions were then examined for similar characteristics to the abnormalities found in the thirty-two fetal and infant individuals from the Kellis 2 cemetery. Similar characteristics include location of the abnormality within the bone, bilateral occurrence, and the age group affected by the pathological condition.

CHAPTER THREE: GROWTH AND DEVELOPMENT

Infant mortality is separated into three categories: late fetal or stillbirth, neonatal, and post-neonatal (Lewis, 2007). Clinically, the first two categories of mortality reflect the endogenous state of the infant as affected by the maternal and genetic influences (Lewis, 2007). The maternal environment plays an important role in the growth and development of the fetus, and the mother's response to social and economic conditions may also have an indirect influence on the fetus (Lewis, 2007). The last category of post-neonatal mortality reflects the exogenous state of the infant as affected by the external environment's influences (Lewis, 2007). It is important to consider the growth and development of the temporal and zygomatic bones to examine the potential contributions of the endogenous and exogenous environments to the conditions considered in this thesis.

Due to the nature of gestational growth and development, congenital malformations, abnormalities, and anomalies can occur. During the gestational period bone growth is very rapid and involves woven or fibrous bone (Ortner, 2003). Various levels of organization, from random to linear, can occur in woven or fibrous bone (Ortner, 2003). Growth in immature bone occurs due to a combination of apposition and resorption, and this modeling process relies upon the adequate supply of nutrients from blood vessels that occurs during vascularization (Baker et al., 2005). Nutritional and endocrine factors influence the onset and rate of bone development and ossification, whether through maternal, placental, or fetal operations (Pryse-Davies et al., 1974). The endocrine system controls the highly regulated process of growth (Lewis, 2007). Because of this, during the prenatal period the fetus is vulnerable to growth retardation (Lewis, 2007). Size and maturity do not necessarily have to advance at the same time even though they are often integrated (Scheuer and Black, 2000).

Anomalies or normal variation can occur in the temporal and zygomatic bones during their growth and development, causing a deviation from the normal appearance of the bone without any functional disturbances (Koesling et al., 2005). Craniofacial anomalies are often caused by mutations in many different genes (Cohen, 2002). Congenital malformations are described as deviations from normal anatomical development and regular function (Koesling et al., 2005). Congenital malformations can be found with advanced, normal, or retarded development of ossification centers (Pryse-Davies et al., 1974). When malformations are discovered, they are often not considered to be of any clinical significance if they represent congenital or developmental anomalies (Epstein and Epstein, 1967).

Malformations could be indications of an underlying pathological cause, and should therefore require more than just a physical analysis to determine a differential diagnosis (Epstein and Epstein, 1967). Anomalies should not be confused with pathological structures (Koesling et al., 2005). Certain diseases can only occur in bone formed by certain processes (Ortner, 2003); for example, certain diseases focus on the skeletal tissue formed from cartilage in endochondral ossification, while others focus on the process involved in membrane bone formation in intramembranous ossification (Ortner, 2003). Bone destruction and osteoclastic activity are associated with acute inflammatory diseases (Ortner, 2003).

The Temporal Bone

The temporal bones articulate with the sphenoid, parietal, occipital, maxillary, and zygomatic bones (Gray, 1977; Scheuer and Black, 2000; Figure 9). Each temporal bone is made up of the petromastoid portion, squamous portion, tympanic portion, and the styloid process (Scheuer and Black, 2000; Figure 10). The squamous portion articulates with the parietal bone and the great wing of the sphenoid, and the petrous portion articulates with the

occipital and parietal bones (Gray, 1977). The squamous portion is thin and translucent, whereas the petrous portion is thick and dense (Gray, 1977). The tympanic portion lies below the squamous portion and anterior to the mastoid process, and the squamous portion fuses posteriorly to the mastoid (Scheuer and Black, 2000). The anterior surface of the petrous portion is joined to the squamous portion by the petro-squamous suture (Gray, 1977). The zygomatic process projects anteriorly from the lower border of the squamous portion (Scheuer and Black, 2000).

Figure 9: Lateral view of adult skull with the location of the temporal bone outlined (AN-0002 European male, courtesy of the Osteology Lab at UCF).

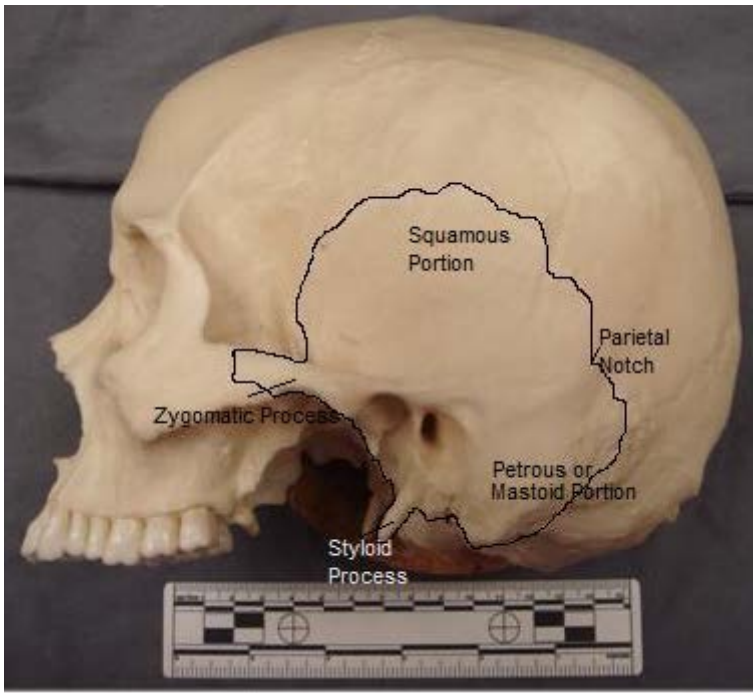


Figure 10: Right adult temporal bone (AN-0002 Adult European male, courtesy of the Osteology Lab at UCF)

The temporal bone is clinically known as an area of difficult anatomy (Koesling et al., 2005). There are fifteen different muscle attachments involving the temporal bone (Gray, 1977). The squamous portion provides attachments for the temporal muscle and the temporal fascia (Gray, 1977). The temporalis muscle covers the lateral surface of the squamous portion of the temporal bone (Scheuer and Black, 2000). The facial and vestibulocochlear cranial nerves and the internal carotid artery pass through the temporal bone (Scheuer and Black, 2000). The apex of the petrous portion contains the internal orifice of the carotid canal, as well as the posterior and external boundary of the foramen lacerum medium (Gray, 1977). Above the termination of the carotid canal there is a shallow depression where the Gasserian ganglion is received (Gray, 1977). The squamoso-petrous sinus, also known as the petrosquamous or petrosquamosal sinus, which carries all the intracranial venous blood in early fetal life before the formation of the internal jugular vein, is found to be present in the temporal bone in fetal life (Cheatle, 1900;

Chell, 1991). However, only parts of the petrosquamous sinus continue to persist into adulthood (Ruiz et al., 2006). When the jugular vein takes over, the hole still remains where the sinus once was (Cheatle, 1900). The petrosquamous sinus, which connects the internal venous sinuses of the dura matter and the external veins of the temporo-mandibular region, runs at an angle between the petrous and squamous portions of the temporal bone (Chell, 1991; Figure 11). The petrosquamous sinus is an emissary vein that occurs bilaterally primarily in the petrous portion of the temporal bone, and is associated with anomalies of venous sinuses or skull base malformations (Chauhan et al., 2011; Table 4). An enlarged mastoid emissary foramen can result in bilateral defects in the lateral petrous portion (Chauhan et al., 2011; Figure 12; Table 4). The petrosquamous sinus has also been identified as having an infra-temporal origin from the mastoid emissary vein (Chauhan et al., 2011).

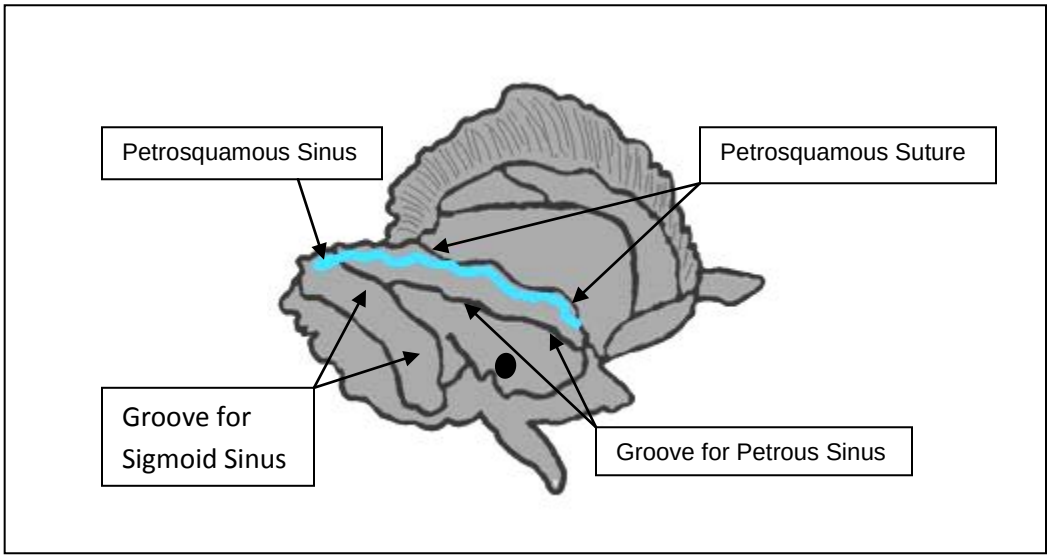


Figure 11: The area in which the petrosquamous sinus (in blue) can occur in the petrosquamous suture on the left adult temporal bone, internal view. The petrosquamous sinus flows through an area of the petrosquamous suture and its emissary is the juncture of the sigmoid sinus (on the internal side) and transverse sinus (on the external side).

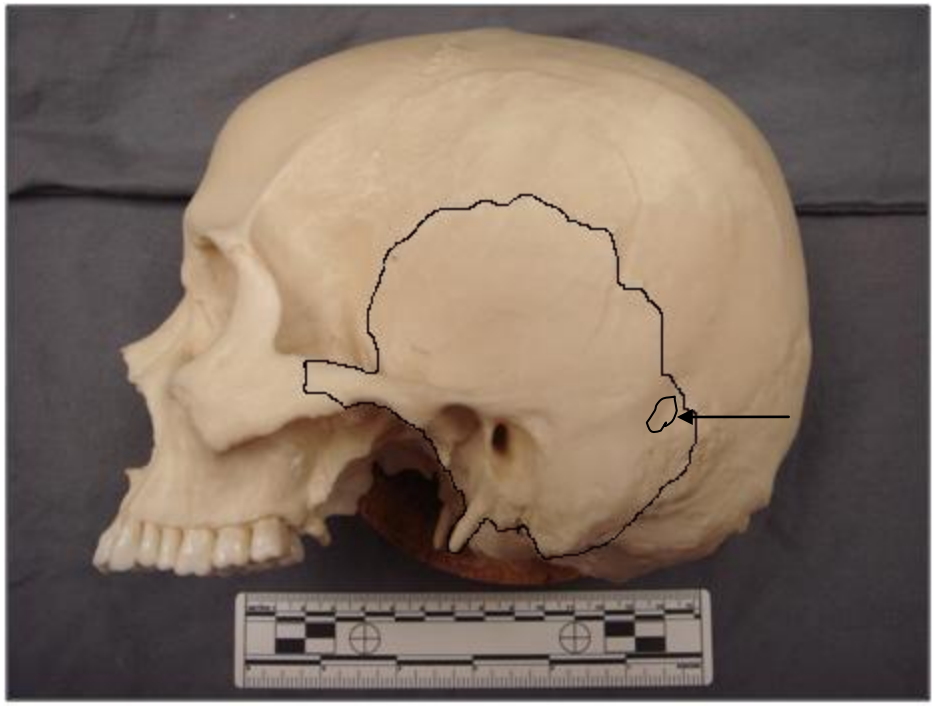


Figure 12: Location of the defect caused by the mastoid emissary foramen if the anomaly occurs. (AN-0002 Adult European male, courtesy of the Osteology Lab at UCF)

The temporal bone forms through a combination of endochondral and intramembranous ossification processes (Lewis, 2007; Baker et al., 2005). Intramembranous ossification occurs earlier than endochondral ossification, and involves the direct mineralization of the membranous template with new bone being deposited on the surface causing the ossification center to expand (Baker et al., 2005). The pars branchialis and pars otica are the two precursors from which the temporal bone develops (Romo et al., 2003). The temporal squama originates in the neural crest (Cohen, 2002), and is considered to be a dermal bone (Wilson, 1955). The other temporal bones have a mesodermal origin (Cohen, 2002). The squamous and tympanic portions form intramembraneously, and the petrous portion pre-forms in cartilage before ossification (Baker et al., 2005). The temporal bone develops from ten ossification centers (Gray, 1977). Ossification centers in the temporal bone appear to arise in groups in relation to the areas of nerve termination, the internal acoustic meatus, and the semicircular canals

(Scheuer and Black, 2000). Most primary ossification centers appear during gestation. The female fetus is more likely to have advanced development of ossification centers (Pryse-Davies et al., 1974). Small-for-dates fetuses and multiple-birth fetuses are more likely to have retarded ossification (Pryse-Davies et al., 1974).

The ossification centers for the pars squama appear during the seventh and eight fetal weeks (Scheuer and Black, 2000). The ossification of the squamous portion of the temporal bone can be seen in CT imaging as early as 17 fetal weeks (Nemzek et al., 1996). The squamous portion of the temporal bone begins with a nearly straight lower edge and becomes a curved process between the tympanic and petrous portion towards the end of gestation (Sutton, 1883).The ossification centers of the tympanic ring fuse at 11 fetal weeks (Krmpotic-Nemanic et al., 1999). The expansion of the tympanic cavity begins the formation of the air cells found in the petrous and mastoid temporal bone, and it reaches adult size by 37 fetal weeks (Nemzek et al., 1996). The osseous labyrinth, auditory ossicles, and tympanic ring of the temporal bone all reach adult proportions during fetal growth, and have no postnatal growth in size (Scheuer and Black, 2000). As the fetal labyrinth grows, the petrous bone re-orientates coronally by about 15 degrees (Jeffery and Spoor, 2004). Mid-fetal life is when both the petrous and the squamous parts become recognizable (Scheuer and Black, 2000; Figure 13). By nine months gestation, the os temporale of the fetus has three ossification centers that have become partially attached but not firmly and not throughout the adjoining borders (Boot, 1910).

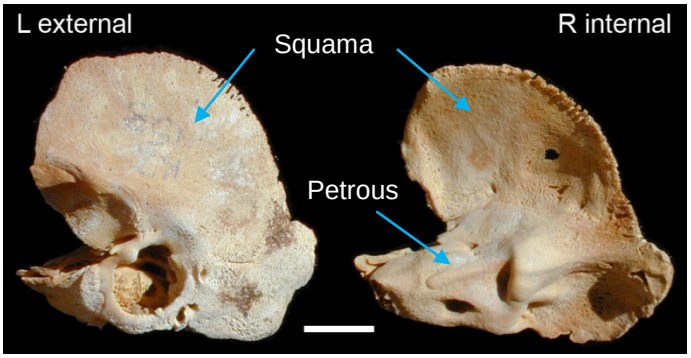


Figure 13: Fetal temporal bones from Burial 153 from Kellis 2, Dakhleh Oasis, Egypt. Estimated age at death is 39 weeks gestation (Wheeler, 2009). (Image courtesy of S. Wheeler)

By the end of fetal life the temporal bone is made up of four parts: the squamo-zygomatic, tympanic plate, petro-mastoid, and styloid process (Gray, 1977). Then, at birth the temporal bone is usually represented by two main parts: the petromastoid and the squamotympanic (Scheuer and Black, 2000). The petrous part of the temporal bone is well ossified by birth (Scheuer and Black, 2000). The neonatal temporal bone has three main parts: the petromastoid, the squamous, and the tympanic ring (Wilson, 1955). The squamo-tympanic part fuses to the petromastoid part of the temporal bone during the perinatal period (Scheuer and Black, 2000). The squamous portion does not fuse to the petrous portion until the first postnatal year (Baker et al., 2005). The petrous part of the temporal bone does not undergo any remodeling, so its primary endochondral bone is retained throughout postnatal life (Scheuer and Black, 2000). However, the mastoid, squamous, and tympanic parts of the temporal bone experience great change in shape and proportion during postnatal life (Scheuer and Black, 2000). In the newborn, the tympanic ring reaches its full size and becomes fixed to the petrous pyramid (Krmpotic-Nemanic et al., 1999). At this time, the pars squama is described as thin and light (Boot, 1910). As the bones come closer together, the sutures became the major locations of intramembranous bony growth during both neonatal and postnatal development (Mesina-Botoran et al., 2007). Only one suture is present in the 9 month old fetus, the petrosquamous

suture, and by birth the petrotympanic suture is present (Boot, 1910). The petrosquamous suture occurs between the squamous and petrous portions (Scheuer and Black, 2000; Figure 5). The petrosquamous suture is a wide cleft at birth, and leads into the tympanum and antrum through the tegmen (Boot, 1910). The petrosquamous sinus can be seen in infancy and early childhood, causing grooves or canals in the location of the petrosquamous suture (Proctor et al., 1981). However, this sinus can be extremely variable in its course (Proctor et al., 1981). The failure of this suture to close leads to dehiscences which cause a predisposition to menigeal and brain infections (Boot, 1910; Table 4).

Abnormalities of the temporal bone are often clinically divided into the external or outer, middle, and inner ear regions (Romo et al., 2003). Abnormalities can occur in the temporal bone without impairing function or imperiling the health of the individual (Romo et al., 2003). Vascular anomalies also exist that can affect the appearance of the temporal bone (Koesling et al., 2005).

Table 4: Developmental abnormalities of the temporal bone.

Condition	Age	Bilateral Occurrence	Appearance	Location on the Temporal Bone	References
Petrosquamous Suture dehiscence	Infant	Yes	Failure of suture to close	Petrous and squamous portion	Boot, 1910; Proctor et al., 1981
Petrosquamous sinus anomalies	Infant	Yes	Enlarged emissary foramen, defect	Petrous portion along the petrosquamous suture	Cheatle, 1900; Chell, 1991; Chauhan et al, 2011; Ruiz et al., 2006
Mastoid emissary vein defect	Infant	Yes	Ovular, enlarged or dilated emissary foramen, defect	Lateral petrous portion, squamous portion	Chauhan et al., 2011

The Zygomatic Bone

The zygomatic bone is also known as the cheek bone, malar bone, or zygoma. These terms, zygoma and malar, are derived from Greek and Latin words for yoke and cheek (Scheuer

and Black, 2000). There are two zygomatic bones of the face, one left and one right. When it comes to fetal cranial bones, the zygomatic bone is considered to be larger and more robust than the other facial bones and therefore is more often recovered (Scheuer and Black, 2000). The bones that articulate with the zygomatic bones are worth noting for those adjacent bones may be primary locations for diseases that spread to include zygomatic involvement. It is also important to know the location of the zygomatic bone for the anatomical features of underlying tissues and vessels that are involved in specific diseases. As part of the facial bones, the right and left zygomatic bones articulate with the maxillae, the greater wings of the sphenoid, and the zygomatic processes of the frontal and temporal bones (Figure 14). The zygomatic bone has an orbital surface which forms part of the floor of the orbit as well as an anterolateral orbital wall (Scheuer and Black, 2000). The zygomatic bone has three projections: the temporal process, the frontal process, and the maxillary process (Baker et al., 2005). The frontal process projects superiorly to meet the zygomatic process of the frontal bone, and the temporal process projects posteriorly to meet the zygomatic process of the temporal bone (Scheuer and Black, 2000). The orbital surface of the zygomatic bone articulates with the greater wings of the sphenoid (Scheuer and Black, 2000). The antero-inferior border of the zygomatic bone is the articulating surface for the maxillary bone (Scheuer and Black, 2000).

Figure 14: Frontal view (left) and lateral view (right) of an adult skull with the location of the zygomatic bones outlined (AN-0002 European male, courtesy of the Osteology Lab at UCF).

Several sutures are involved with the zygomatic bone: the zygomatic-maxillary suture, the anterior limb of the coronal suture system, and the zygomatico-frontal suture (Scott, 1953). The location of the zygomatic bone amongst the facial bones creates growth tensions so that the basi-occipital synchondrosis can separate the zygomatic bone from the temporal bone at the zygomatic arch and can also draw the zygomatic bone backwards along with the temporal bone (Scott, 1953). The anterior limb of the coronal suture system forms the suture between the zygomatic bone and the greater wing of the sphenoid within the orbital cavity (Scott, 1953). Growth at the zygomatic-maxillary suture can cause the maxillary bone to be thrust inwards, or thrust the zygomatic bone outwards (Scott, 1953).

The zygomaticofacial foramen is located on the facial surface of the body of the zygomatic bone (Baker et al, 2005). The zygomaticofacial foramen perforates the surface of the zygomatic bone near its center, and allows the zygomaticofacial nerve and vessels to pass

through (Gray, 1977). The facial nerve is also anatomically close to the zygomatic bones (Gray, 1977). The zygomatic bone is generally believed to ossify from three centers, with one center for the malar portion and two for the orbital portion (Gray, 1977). These three ossification centers appear around the eighth gestational week, becoming fused around the fifth month of gestation (Gray, 1977). The zygomatic bone is sometimes described as having a horizontal suture present after birth that divides the bone into a large upper portion and a smaller lower portion (Gray, 1977).

Muscles have attachments and origins on the zygomatic bones. A slight elevation below the zygomaticofacial foramen provides the origin for the zygomaticus (Gray, 1977). The zygomaticus major and zygomaticus minor cross the zygomatic bones and aid in facial expression (Gray, 1977). The masseter muscle is attached to the postero-inferior border of the zygomatic bone (Gray, 1977). This border is also referred to as the masseteric border, and is often roughened from the attachment to the masseter muscle (Scheuer and Black, 2000). The temporal fascia attaches to the postero-superior or temporal border of the zygomatic bone (Gray, 1977).

Ossification can be seen in the zygomatic bone by the end of the embryonic period (Baker et al., 2005). The zygomatic bone has its origins in the mesenchyme, and its primary ossification center appears as triangular and squamous or flat around the eighth week of fetal development (Scheuer and Black, 2000). By the ninth week of intrauterine life, the zygomatic bone already begins to resemble an adult zygomatic bone in its basic shape, and by the end of the first half of prenatal life it easily recognizable though proportionally smaller than the adult version (Scheuer and Black, 2004; Figure 15). The temporal process is the first to develop from the squamous portion, followed by the frontal and maxillary processes (Baker et al., 2005). By

sixteen gestational weeks, the zygomatic process can be seen in CT imaging (Nemzek et al., 1996). The temporal process of the zygomatic bone does not completely create the zygomatic arch until late in the intrauterine period or sometimes not until after birth (Scheuer and Black, 2004).

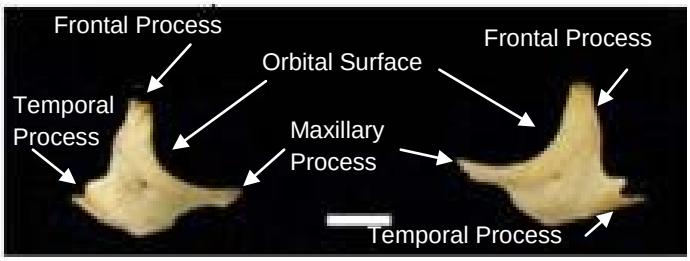


Figure 15: Fetal zygomatic bones from Burial 686 from Kellis 2, Dakhleh Oasis, Egypt. Estimated age at death is around 6 months of infancy (Wheeler, 2009); external view of both right and left zygomatic bones. (Image courtesy of S. Wheeler)

During childhood growth, the temporal process becomes increasingly thicker than the fetal version (Baker et al., 2005). In infancy and early childhood, the growth of the zygomatic bone develops at a rate to keep up with the growth of the maxilla, which is rapidly expanding to accommodate deciduous dentition (Scheuer and Black, 2000). The malar tubercle, which is often seen in adult bones, is not present in fetal and neonatal bones because it is a secondary sexual characteristic that does not become notable until puberty (Scheuer and Black, 2000). Adult proportions of the zygomatic bone are not achieved until about two to three years of age, when the temporal and frontal processes also become serrated (Scheuer and Black, 2000).

CHAPTER FOUR: PATHOLOGICAL CONSIDERATIONS

Pathological conditions such as disease and traumatic injury can result in lesions when they involve bone. Previous studies on the skeletal pathology from the Kellis 2 cemetery have reported evidence of conditions such as periostitis, porotic hyperostosis, benign and malignant neoplasms, osteogenesis imperfect, congenital neural tube defects, and leprosy (Cope, 2008; Cope and Dupras, nd; Mathews, 2008; Molto, 2001; Molto, 2002; Wheeler, 2009). Previous studies have also found evidence of trauma in the skeletons of the Kellis 2 population including fractures and humerus varus deformity (Molto, 2002; Wheeler, 2009). Non-specific indicators of stress have also been found in the population at Kellis 2, such as cribra orbitalia and enamel hypoplasias (Molto, 2002; Wheeler, 2009). Although these previous studies have covered lesions manifesting throughout the skeleton, this study the focus will be solely on the differential diagnosis of those pathological conditions that manifest in the temporal and malar bones of fetuses and infants.

When creating a differential diagnosis, the gross examination of the abnormality or defect provides critical information based upon where the abnormality occurs, its appearance, and the age and sex of the individuals in which it has been found to occur. In this sample, the location of the defect or lesion is the temporal bone and the zygomatic bone. There is bilateral occurrence for both the temporal bone defects and the zygomatic bone lesions. Sex may be an important factor for increasing and decreasing the likelihood of the abnormality in question being the result of certain conditions (Ortner, 2003), but the fetal and neonatal age range of this sample prevents an estimation of sex. Knowing the age at death of the individual is also important when performing a differential diagnosis because the age increases or decreases the likelihood of occurrence of certain conditions (Ortner, 2003). A condition is considered to be

congenital if it is present at birth, but can be differentiated as either hereditary or acquired depending upon whether the causal factors of the condition were present in the zygote or obtained during gestational development (Schuknecht, 1993). Hereditary conditions can be due to genetic abnormalities such as chromosomal disorders, single gene mutations, and polygenic inheritance (Schuknecht, 1993). Congenital anomalies of the skeleton are the result of pathological changes in the normal development that occurs during the gestational period (Aufderheide and Rodriguez-Martin, 1998). Congenital anomalies that affect the growth and development of the skeleton generally leave more unmistakable evidence than inflammatory bone lesions (Putschar, 1966). Therefore, conditions that manifest in the temporal bone will be examined for occurrence in fetal and neonatal age groups and for bilateral occurrence, and whether or not the lesion manifests in the squamous and petrous portion of the temporal bone. Likewise, the same will be done for the zygomatic bone. The conditions have been divided into general infections, bone diseases and neoplasms, systemic diseases, and metabolic diseases. Descriptions of the abnormalities associated with these conditions will also be provided for later comparisons to the defects and lesions found in this sample.

Infections

Infections can be caused by a bacteria, virus, fungus, or parasite (Aufderheide and Rodriguez-Martin, 1998). In most cases, it is difficult to tell what type of infection led to the presence of periostitis or osteomyelitis, but some bacterial causes have specific types of lesions and/or distribution of lesions that allow for specific identification in the skeleton (Mays, 2002).

Infections of the Temporal Bone

Infections of the temporal bone are clinically categorized by region: external ear, middle ear and mastoid, inner ear, and petrous apex (Nemzek and Swartz, 2003). The cycle of

pathological conditions has a focal point from which the disease radiates or spreads (Goldstein, 1912). In the clinical setting, infections of the temporal bone are seen most commonly beginning in the middle ear and Eustachian tube, eventually spreading to other areas as the infection grows (Graham-Hodgson, 1950). After the Eustachian tube, infections tend to spread to the petrous bone, middle ear, and lastly to the mastoid (Graham-Hodgson, 1950). As the petrous pyramid grows, the fibroblastic marrow is replaced by an air-filled mucous membrane that pushes into the antrum, mastoid, and then into the squamous portion (Fowler, 1940). The tympanic cavity is highly susceptible to infection due to its connection with the upper respiratory tract (Goldstein, 1912). The various emissary veins of the lateral sinus can carry infection to other portions of the temporal bone (Goldstein, 1912).The mastoid and the petro-mastoid is therefore often the site of secondary infections (Graham-Hodgson, 1950). Poorly pneumatized or infantile mastoids have a higher incidence of infection (Graham-Hodgson, 1950). Mastoiditis can lead to the formation of abscesses, with one or more of the walls of the cells breaking down (Graham-Hodgson, 1950; Table 5). The squamous and petrous portions of the temporal bone can be involved in mastoiditis (Johnson, 1940). Besides lesions, infection can also cause increased density in the petrous bone when viewed radiographically on the side that the infection is present (Graham-Hodgson, 1950). Infection can occur in one or both temporal bones (Graham-Hodgson, 1950).

One type of infection that affects the temporal bone is otitis media, an infection in which infants are highly susceptible (Fowler, 1940; Table 5). When in the air cells of the temporal bone, the infection of otitis media will sometimes burst through the cortex and can lead to osteomyelitis (Fowler, 1940). Infections in the middle ear can lead to bony involvement of the petrous pyramid, with effects such as osteomyelitis, the breakdown of cell partitions, and sequestrations (Lindsay, 1945). Infection that occurs in the mastoid is an infection of the air cell

system and is not an osteomyelitis (Lindsay, 1945). Infection generally spreads along pneumatized tracts from the middle ear to the petrous portion (Lindsay, 1945).

Osteomyelitis occurs most often as a result of the introduction of pyogenic bacteria into bone, but can also occur as a result of infectious agents that enter the bone directly from a wound, by direct extension from soft tissue adjacent infections, or by a hematogenous route from a remote septic focus (Ortner, 2003). When infection is limited to the periosteum and cortex and not spread through the medullary cavity, there is often a focal periosteal bone deposition around a cortical defect, sometimes occurring with a small sequestrum or some sclerotic activity (Ortner, 2003). When these infections heal, sclerotic scarring can occur around a depression, and may later be effaced by remodeling (Ortner, 2003). Streptococcic osteomyelitis is a disease that occurs mostly in children and can occur bilaterally in the temporal bone (Boyd-Snee, 1923; Table 5). It is a bacterial disease involving an acute inflammatory reaction that primarily affects the tympanic portion of the temporal bone, though the condition can spread and involve other portions of the temporal bone (Boyd-Snee, 1923). Streptococci are twice as likely as staphylococci to be responsible for the infection (Aufderheide and Rodriguez-Martin, 1998). Although found in infants of all ages, this condition is not a congenital condition. Acute hematogenous osteomyelitis is found in infants who are pre-disposed to the condition from factors such as pre-eclampsia, premature rupture of membranes, and local infections (Aufderheide and Rodriguez-Martin, 1998).

Table 5: Infections in the temporal bone

Condition	Age	Bilateral Occurrence	Appearance	Location on the Temporal Bone	References
Mastoiditis	Infants	Yes	Abscesses, one or more walls broken down, increased density	Squamous, petrous portions; mastoid	Graham-Hodgson, 1950; Johnson, 1940
Otitis media	Infants	No	Osteomyelitis, breakdown of cell partitions, sequestrations	Spreads from middle ear to petrous portion	Fowler, 1940; Lindsay, 1945
Streptococcic osteomyelitis	Infants and children	Yes	Acute inflammatory reaction	Spreads from tympanic portion	Ortner, 2003; Boyd-Snee, 1923; Aufderheide and Rodriguez-Martin, 1998

Infections of the Zygomatic Bone

Congenital syphilis transmits from the mother to the fetus through the placenta after about sixteen to eighteen weeks gestation (Aufderheide and Rodriguez-Martin, 1998). Congenital syphilis can result in fetal death during pregnancy, a stillborn fetus at birth, or a delivered living infected infant (Ortner, 2003). Those early-term fetal deaths do not display skeletal manifestations, but the two latter categories do display bony changes usually in the form of syphilitic osteochondritis (Ortner, 2003; Table 6). The skeletal lesions of congenital syphilis appear in the form of osteochondritis in areas where endochondral ossification takes place (Aufderheide and Rodriguez-Martin, 1998). However, the primary locations for the spirochetes to infect in newborns and infants are the metaphyseal tissues (Aufderheide and Rodriguez-Martin, 1998). Skull lesions are rarely seen in congenital syphilis, and when they are present they are mostly hypertrophic periostitis or necrotizing osteitis (Aufderheide and Rodriguez-Martin, 1998). Lesions are most commonly found in the distal femur and proximal tibia due to their fast growing metaphyses, but lesions can be found throughout the entire infant skeleton (Ortner, 2003). During infancy, syphilitic periostitis forms, though it also can begin forming intrauterine (Ortner, 2003). Early congenital syphilis appears most often in the skull,

nasal bones, and long bones in infants (Khurana and Fitzpatrick, 2009). Early congenital syphilis needs to be differentiated from rickets and scurvy (Khurana and Fitzpatrick, 2009). Congenital syphilis manifests as a bone infection in skeletal remains (Anderson, 2000). Often, by a few months of age reactive periosteal new bone and healing has occurred (Khurana and Fitzpatrick, 2009). As the infant grows into a child, active syphilitic changes of the bone occur and begin to resemble acquired syphilis until adolescence when it becomes nearly impossible to differentiate the two forms of syphilis (Ortner, 2003). When present in the skull, lesions of congenital syphilis appear as rounded with destructive foci and multiple locations (Ortner, 2003). The involvement of facial bones can occasionally occur in congenital syphilis, and can also have extensive manifestations (Ortner, 2003). Lesions with new bone formation have been identified in the zygomatic bones of infants and children, though these lesions are considered to have a differential diagnosis of both congenital syphilis and yaws (Buckley and Tayles, 2003).

Tuberculosis is caused by an infection of an organism. The acute or chronic infections of both the soft and skeletal tissues in tuberculosis are caused by *Mycobacterium tuberculosis* or *M. bovis* (Aufderheide and Rodriguez-Martin, 1998). Tuberculosis is generally a biphasic disease, with a primary infection phase of the lungs followed by a re-infection phase where the infection is disseminated through the blood stream to any and all organs (Aufderheide and Rodriguez-Martin, 1998). Tuberculosis osteomyelitis affects the bone more often in children than in adults (Rothschild and Martin, 2000; Table 6), and in children, the cranial vault is the most common location in the skull, followed by the facial bones, and very rarely occurs in the cranial base (Ortner, 2003). Tuberculosis of the flat bones of the face is considered rare clinically, and more commonly skeletal symptoms of tuberculosis occur in the long bones and vertebral column (Meher et al., 2003). Even rarer is when the primary site of tuberculosis is in the facial bones (Meher et al., 2003). Thus, zygomatic tubercular infection is considered

extremely rare (Meher et al., 2003). However, involvement of the zygoma and the junction of the zygoma and the maxilla are considered common in small children with tuberculosis (Ortner, 2003).

Yaws is another form of treponemal disease referred to scientifically as *Treponema pertenue,* that is nonvenereal and can often have similar effects as the venereal form (Buckley and Tayles, 2003). Yaws generally occurs early in life, increases in toddlers, and continues throughout childhood (Buckley and Tayles, 2003; Table 6). In a case study on a prehistoric Pacific Island population, pathologies were found on the facial bones of adolescents including the nasal area, superior aspect of the palate, and the zygomatic bones (Buckley and Tayles, 2003). The lesions of yaws are similar to those of congenital syphilis, but without osteochondritis (Ortner, 2003). Yaws has not been identified in the neonatal age group or younger (Ortner, 2003). Chronic lesions of the skull are described as a central crater-like destruction surrounded by reactive bone formation, though they may begin as a roughly circular cluster of holes penetrating the outer table (Ortner, 2003).

Table 6: Infections of the zygomatic bone

Condition	Age	Bilateral Occurrence	Appearance	Location on the Zygomatic Bone	References
Congenital syphilis	Fetal, Infant, Children	Multifocal	Rounded with destructive foci, hypertrophic periostitis, necrotizing osteitis	External surface	Aufderheide and Rodriguez-Martin, 1998; Ortner, 2003; Khurana and Fitzpatrick, 2009
Tuberculosis	Children	Multifocal	Osteomyelitis	Junction of zygoma and maxilla	Aufderheide and Rodriguez-Martin, 1998; Rothschild and Martin, 2000; Ortner, 2003; Meher et al., 2003
Yaws	Infants, Children	Multifocal	Roughly circular cluster of holes, then central crater-like destruction surrounded by reactive bone formation	External surface	Buckley and Tayles, 2003

Systemic Diseases

Systemic diseases such as histiocytosis x, unifocal eosinophilic granuloma, Hand-Schueller-Christian disease, and Letterer-Siwe disease all can have manifestations in the temporal bone (Nadol and Merhant, 1993). Certain neoplastic diseases, such as multiple myeloma, leukemia, metastic tumors, and paraganglioma, have systemic implications as well as manifestations in the temporal bone (Nadol and Merhant, 1993). Neoplastic conditions will be discussed later on in this chapter. Myeloma is a highly malignant disorder of plasma cells that generally arises in the hematopoietic bone marrow, occurring first as a solitary site and quickly progressing to multiple sites (Ortner, 2003). Many areas of the skeleton can be affected by myeloma, and skeletal involvement is a common manifestation of myeloma (Ortner, 2003). The lesions of myeloma often penetrate both tables of the skull, and often have scalloped margins

(Ortner, 2003). Myeloma is the most common of the white blood cell disorders and rarely occurs in modern medicine in individuals under the age of forty (Aufderheide and Rodriguez-Martin, 1998). Tumors rarely occur before 40 years of age in myeloma (Ortner, 2003). Lesions with sharply demarcated edges occur on the skull and complete destruction of the osseous tissue within the perimeter of the lesion, and lack reactive new bone formation can also occur (Aufderheide and Rodriguez-Martin, 1998). Multiple myeloma is known for its punched-out osteolytic lesions (Nadol and Merhant, 1993). Histiocytosis X has very similar bone lesions among its three different clinical manifestations (Ortner, 2003). In all three conditions there is a proliferation of histiocytes in the organs and tissues (Ortner, 2003). In general, the lesions caused by histiocytosis X are round or oval and have an undulating border if they coalesce (Ortner, 2003).

Systemic Diseases of the Temporal Bone

Langerhans' cell histiocytosis most commonly occurs in the temporal bone with bilateral temporal bone involvement though sometimes occurring unilaterally (Fernandez-Latorre et al., 2000; Table 7). Langerhans' cell histiocytosis presents clinically as extensive lytic lesions associated with a soft tissue mass in pediatric patients (Fernandez-Lattore et al., 2000). The peak age range of incidence of Langerhans' cell histiocytosis is between 1 and 4 years of age (Saliba et al., 2008). Bilateral bone destruction occurs in the squamous, petrous, and mastoid portions of the temporal bones (Fernandez-Lattore et al., 2000). Radiological presentations of Langerhans' cell histiocytosis show the appearance of lytic lesions that are diffusely destructive, or as a destructive bony lesion when using computerized tomography (Saliba et al., 2008). Letterer-Siwe disease is the most malignant form of Langerhans' cell histiocytosis (Balakrishnan et al., 1997). Letterer-Siwe disease is a chronic and most often fatal condition involving skeletal and extra-skeletal tissues that occurs in children less than two years old (Balakrishnan et al.,

1997; Table 7). Letterer-Siwe disease can have bilateral temporal bone involvement, and has been documented in the mastoid, petrous, and squamous portions (Balakrishnan et al., 1997). However, this disease presents with osteolytic lesions that more commonly occur on the other flat bones of the skull and long bones (Balakrishnan et al., 1997). Letterer-Siwe disease generally manifests as multiple bone lesions of the cranial vault and base, with a higher rate of occurrence on the sphenoid and a lesser rate of occurrence on the facial bones (Ortner, 2003). Eosinophilic granuloma is the most common form of histiocytosis X, and is characterized by single or multiple lesions in children and young adults (Ortner, 2003). Eosinophilic granuloma very rarely has been clinically found to involve the temporal bone, but when it does it has been found to occur bilaterally in infants (Yetiser et al., 2002; Table 7). This condition involves histiocytosis-X system like Letterer-Siwe Disease and Hand-Schueller-Christian Disease (Yetiser et al., 2002). Eosinophilic granuloma lesions are destructive inflammatory granulomas of both the soft tissue and skeleton that occur more frequently in the flat bones of the skeleton (Yetiser et al., 2002). In eosinophilic granuloma, the general manifestation of the lesion is solitary, purely lytic, round or oval, and has a beveled edge (Ortner, 2003). The most common location for a lesion of eosinophilic granuloma is the cranial vault (Ortner, 2003).

Table 7: Systemic diseases of the temporal bone

Condition	Age	Bilateral Occurrence	Appearance	Location on the Temporal Bone	References
Langerhans' cell histiocytosis	Infants and young children; 1-4 years of age	Yes	Extensive, destructive lytic lesions	Squamous, petrous, and mastoid portions	Fernandez-Latorre et al., 2000; Saliba et al., 2008
Letterer-Siwe disease	Infants; less than 2 years of age	Yes	Osteolytic lesions	Mastoid, petrous, Squamous portions	Balakrishnan et al., 1997; Ortner, 2003
Eosinophilic granuloma	Infants, children and young adults	Yes	Purely lytic, beveled edge, rounded or oval, destructive granuloma lesions of soft tissue and skeleton	Mastoid	Ortner, 2003; Yetiser et al., 2002

Systemic Diseases of the Zygomatic Bone

Hand-Schuller-Christian disease generally manifests with large, multiple, confluent defects (Ortner, 2003; Table 8). When these lesions occur on the cranial vault, they are usually devoid of periosteal reaction even after both tables have been destroyed (Ortner, 2003). Bony involvement often includes the mastoid bone, zygomatic bone, tympanic bone, and sphenoidal bone (Saliba et al., 2008). This disease has been identified in infants and children, though bilateral occurrence is rare (Saliba et al., 2008).

Table 8: Systemic disease of the zygomatic bone

Condition	Age	Bilateral Occurrence	Appearance	Location on the Zygomatic Bone	References
Hand-Schuller-Christian disease	Infants, Children	Yes	Defects with destruction of both tables w/o periosteal reaction	Orbital surface	Ortner, 2003; Saliba et al., 2008

Metabolic Diseases

Metabolic diseases are also referred to as indicators of stress in paleopathology (Roberts and Manchester, 2005). Metabolic bone disease refers to any conditions that have specifically disrupted the process of bone modeling and remodeling, and these conditions do not cause changes that affect the entire skeleton (Brickley and Ives, 2008). The development of a metabolic bone disease involves a multitude of factors such as sex, age, environment, nutrition, culture, exposure, and host resistance (Brickley and Ives, 2008). Metabolic bone diseases include many conditions that result from disorders of bone resorption and/or formation, which often affected the mineral or matrix components (Khurana and Fitzpatrick, 2009). A strong relationship occurs between infectious disease and nutritional status, and nutritional status is affected by metabolic disease, as a person with low nutritional status has a lowered immunity to infectious diseases (Roberts and Manchester, 2005).

Metabolic and endocrine conditions cause changes in both soft tissue and the skeleton as a result too little or too much of a certain hormone being produced (Aufderheide and Rodriguez-Martin, 1998). Fetal calcium and bone metabolism is uniquely able to meet the specific developmental needs in order to fully mineralize the skeleton (Kovacs, 2003). During the gestational period, the blood calcium levels of the fetus are about equal to those of the mother, and then fetal levels drop about 20% after delivery (Kovacs, 2003). Abnormal levels whether high or low cause a mineral imbalance during the development of the fetal skeleton (Kovacs, 2003).

Infants become at risk for developing metabolic bone disease when they have an inadequate supply of phosphorous and calcium (Bishop and Fewtrell, 2003). This occurs especially in preterm infants, who are more likely to have a low bone mass (Bishop and

Fewtrell, 2003). Preterm infants, at around 37 weeks gestation, are born during a rapid phase of mineral accretion and as a result have much lower mineral stores than full term infants (Bishop and Fewtrell, 2003). Mineral deficiency results in abnormal bone remodeling, reduced linear growth and low bone mass (Bishop and Fewtrell, 2003). Clinically, signs of mineral deficiency begin to appear relatively late in the neonatal period; these signs are considered to be unusual and usually only found by accident when x-rays are being taken for other reasons (Bishop and Fewtrell, 2003). In societies where adequate nutrition and exposure to sunlight occurs, vitamin D deficiency is uncommon (Ortner, 2003). Calcium disorders that are only minimally or not at all affected by dietary vitamin D and can therefore still occur even when there is no vitamin D deficiency (Ortner, 2003).

Metabolic Diseases of the Zygomatic Bone

The clinical manifestation of vitamin C deficiency is called scurvy, and the vitamin deficiency can cause skeletal changes (Roberts and Manchester, 2005; Table 9). This is because the lack of vitamin C causes a defective collagen and osteoid synthesis, which in turn results in skeletal growth retardation and hemorrhagic phenomena (Aufderheide and Rodriguez-Martin, 1998). Infants cannot synthesize Vitamin C and instead depend on the mothers to produce breast milk that contains synthesized Vitamin C (Brickley and Ives, 2006). Children are more susceptible than adults in becoming symptomatic when their vitamin C levels are deficient (Aufderheide and Rodriguez-Martin, 1998). In fact, the skeletal evidence of scurvy is highest during infancy and early childhood (Brickley and Ives, 2006). Children can develop the symptoms of scurvy faster than in adults, and most commonly is found in infants between 2-24 months of age (Brickley and Ives, 2006). Clinically, scurvy is most often found during infancy between about 5-10 months of age (Khurana and Fitzpatrick, 2009). The primary symptom of scurvy is the appearance of hemorrhages, which occur in the skin and soft tissue as well as the

joints and bones (Aufderheide and Rodriguez-Martin, 1998). Individuals undergoing an active growth period are most likely to manifest the most adverse effects of scurvy (Ortner et al., 1999). In children, growth occurs faster along with rapid remodeling, which results in the increased vulnerability to hemorrhage and the possibility of inflammatory responses (Ortner and Ericksen, 1997). Lesions on the skull attributed to scurvy have been observed for over a hundred years (Ortner and Ericksen, 1997). In the skull, scurvy is represented by porous lesion as well as the enlargement or thickening of bone tissue (Ortner and Ericksen, 1997). Skeletal manifestations of scurvy can be found in the greater wings of the sphenoid and adjacent bone tissue as well as the orbital roof (Ortner and Ericksen, 1997). Sometimes the lesions of the skull have a related thickening of the zygomatic region (Brown and Ortner, 2011). In archaeological human remains, scurvy has not been well documented in infants and children (Ortner et al., 1999). A scarcity of cases of infantile scurvy exists within the archaeological record probably due to a lack of understanding and recognition of the manifestations of scurvy on the bone (Brickley and Ives, 2006). This is due most likely to the variable presentation of scurvy, its representation in forms that are commonly attributed to other conditions like anemia, and the fact that not all children who had scurvy died as children (Ortner et al., 1999). In the archaeological cases, the presence of lesions on the zygomatic bones is considered a typical location (Mays, 2008). Dense, porous, abnormal bone growth has been described in the zygomatic bones of an infant, occurring bilaterally and almost symmetrically (Brown and Ortner, 2011). The internal and external surfaces also showed increased abnormal porosity, especially near foramen (Brown and Ortner, 2011).

Table 9: Metabolic disease of the zygomatic bone

Condition	Age	Bilateral Occurrence	Appearance	Location on the Zygomatic Bone	References
Scurvy	Infants, Children	Yes, Multifocal also	Hemorrhagic, inflammatory response, porous lesion, enlargement or thickening of bone tissue; increased porosity; new bone formation	Orbital surface, external and internal surfaces	Roberts and Manchester, 2005; Aufderheide and Rodriquez-Martin, 1998; Brickley and Ives, 2006; Khurana and Fitzpatrick, 2009; Ortner and Ericksen, 1997; Ortner et al., 1999; Mays, 2008; Brown and Ortner, 2011

Bone Diseases and Neoplasms

A neoplasm is classified as a mass of localized tissue growth that is unregulated by normal growth mechanisms (Aufderheide and Rodriguez-Martin, 1998). Neoplastic disease can either be caused by exposure to carcinogens or by heritability (Aufderheide and Rodriguez-Martin, 1998). Neoplasms can be qualified as either benign and having some physiological restraints, or malignant and having no physiological restraints and therefore able to destroy surrounding normal tissues and spread through the body to create new growth sites (Aufderheide and Rodriguez-Martin, 1998). Tumors can be of a soft tissue origin and cause damage to adjacent skeletal tissue, or be of an osseous origin and have a more direct effect on the skeletal tissue (Aufderheide and Rodriguez-Martin, 1998). Neoplasms of the bone generally present with a sunburst pattern of spicules that are arranged perpendicular to the bone's surface (Aufderheide and Rodriguez-Martin, 1998). Benign tumors have sharply demarcated

peripheral borders, and only affect surrounding areas by compression (Aufderheide and Rodriguez-Martin, 1998). Malignant tumors often have tentacular projections that obscure their peripheral borders, and create room for expansion through necrosis of surrounding cells (Aufderheide and Rodriguez-Martin, 1998). Most of the cases of neoplastic disease in the archaeological record involve adult skeletal material (Aufderheide and Rodriguez-Martin, 1998).

Bone Diseases and Neoplasms of the Temporal Bone

Bone diseases found in the temporal bone such as Paget's disease, osteogenesis imperfecta, and osteopetrosis sometimes mimic otosclerosis (Nadol and Merhant, 1993). Paget's disease, or osteitis deformans, is both osteolytic and osteoblastic (Nadol and Merhant, 1993). Paget's disease usually does not show any clinical manifestations until the sixth decade of life (Nadol and Merhant, 1993). Osteogenesis imperfecta is characterized by bone fractures that occur throughout the skeleton, even on the temporal bone (Nadol and Merhant, 1993). Fibrous dysplasia of the temporal bone is represented by bony deformities and pathologic fractures (Nadol and Merhant, 1993). Osteopetrosis is a rare dysplasia that is characterized by greatly increased bone density, and can be found in the temporal bone accompanied by calcified cartilage (Nadol and Merhant, 1993). Osteitis is characterized by the osteoclastic resorption of bone, fibrosis of marrow, bone cysts, and fractures, and is clinically considered to manifest only very rarely in the temporal bone (Nadol and Merhant, 1993).

Arachnoid granulations appear as scalloped-edged dural defects on the posterior wall of the temporal bone (Lee et al., 2008; Table 10). Arachnoid granulations are pseudopodial projections, but they can cause cortical bone erosion and CSF leakage (Lee et al., 2008). These defects have been found clinically through CT imaging in infants, though they are more

commonly found in adults when the defects become more prevalent and developed (Lee et al., 2008). These defects rarely occur bilaterally (Lee et al., 2008).

Cholesteatoma has both acquired and congenital forms, and is a non-neoplastic destructive lesion (Persaud et al., 2007; Table 10). Cholesteatoma may occur in the inner or outer ear, and it originates as a collection of dead epithelial cells that become infected with pathogenic organisms (Mays and Holst, 2006). Congenital cholesteatoma occurs when aberrant ectoderm becomes trapped during embryogenesis, and can occur anywhere in the temporal bone (Schmalfuss, 2006). Cholesteatoma can also cause the erosion of the surrounding temporal bone (Mays and Holst, 2006). The surrounding bone tends to experience resorption due to this type of lesion (Mays and Holst, 2006). The resorptive lesions have slightly sclerotic margins (Mays and Holst, 2006). The areas near the oval and round windows are the most common locations for lesions of otosclerosis (Mays and Holst, 2006). Bilateral occurrence in the temporal bones is considered typical of otosclerosis (Mays and Holst, 2006). Middle ear cholesteatoma may be congenital or acquired, though the former is the minority (Mays and Holst, 2006). In congenital middle ear cholesteatoma, an expanding mass is seen behind the tympanic membrane (Mays and Holst, 2006). Congenital middle ear cholesteatoma only occurs bilaterally in about 3% of clinically cases (Mays and Holst, 2006).

Metastic neoplasms have been found on the petrous apex of the temporal bone, and are destructive and osteolytic (Nadol and Merhant, 1993). Rhabdomyosarcoma occurs mainly in childhood and less often in adolescence, and is found on the head and neck in 40% of clinical cases (Freling et al., 2010; Table 10). Embryonal rhabdomyosarcoma is a highly aggressive soft tissue tumor found mostly in infants and children that can have temporal bone involvement (Viswanatha, 2007; Table 10). Embryonal rhabdomyosarcoma has a mesenchymal origin, and

manifests first in the middle ear and then spreads to the mastoid and petrous portions (Viswanatha, 2007). This tumor can present in osteo-lytic metastases, also referred to as bone metastases (Freling et al., 2010). When occurring within one of the facial bones, rhabdomyosarcoma can cause bony changes in the surrounding bones as the tumor spreads, including the zygomatic bones (Freling et al., 2010). Primary sites do not include the zygomatic bones, but the primary sites of the orbit and the nasal sinuses places the zygomatic bones in the path of expansion (Freling et al., 2010).

Benign neoplasms or tumors also occur in the temporal region. A type of benign neoplasm called exostosis commonly occurs bilaterally, and manifests as broad-based lesions along the tympanomastoid or tympanosquamous suture lines (Nemzek and Swartz, 2003; Table 10).

Table 10: Bone diseases and neoplasms of the temporal bone

Condition	Age	Bilateral Occurrence	Appearance	Location on the Temporal Bone	References
Arachnoid granulations	Infants	Yes	Scalloped-edged dural defects, cortical bone erosion	Posterior wall	Lee et al., 2008
Cholesteatoma (acquired and congenital)	Fetal, Infant	Yes	Non-neoplastic destructive lesion, surrounding bone erosion, resorption with slightly sclerotic margins	Anywhere	Persaud et al., 2007; Mays and Holst, 2006; Schmalfuss, 2006
Rhabdomyosarcoma	Fetal, Infant	No	Destructive, osteolytic, metastases, bony changes in surrounding bones as it spreads	First in middle ear, then spreads to mastoid and petrous portions	Freling et al., 2010; Viswanatha, 2007
Exostosis	Children	Yes	Broad based lesions	Along the tympanomastoid, tympanosquamous suture lines	Nemzek and Swartz, 2003; Kern and Macdonald, 1961

Bone Diseases and Neoplasms of the Zygomatic Bone

Myofibroma is a rare disease of benign modules in skin, muscle, and bone; orbital involvement is the rarest form of myofibroma (Nam et al., 2005; Table 8). One clinical case study involves a three year old male with infantile myofibroma presented with a mass on the orbital surface of the zygomatic bone, and the mass had caused erosion of the bone (Nam et al., 2005). Computed tomography scans showed this case of infantile myofibroma to be a well circumscribed mass with orbital extension/protrusion (Nam et al., 2005). On a computed tomography with enhanced contrast, the isolated lesions of bone tend to be round and well-defined and surrounded by sclerotic rims of bone (Nam et al., 2005).

One type of osseous tumor is intraosseous hemangioma, which represents less than 1% of osseous tumors in clinical data (Moore et al., 2001; Table 8). Hemangioma is a type of lesion that is very rare in any part of the skeleton, and is a neoplasm caused by proliferating blood vessels (Ortner, 2003). Hemangiomas are considered to be a common primary bone lesion (Koulouris and Rao, 2005). Hemangiomas are usually round lesions of several centimeters in diameter that cause the destruction of the inner and outer table and outward expansion causes a circular lytic margin around the peripheral edges (Ortner, 2003). Only twenty clinical cases of intraosseous hemangioma are known to be located in the zygoma (Moore et al., 2001). However, patients usually present with this condition in the fourth decade of life (Moore et al., 2001). Hemangiomas are clinically the most common tumors found in infants, including preterm infants (Waner and Suen, 1999). Bony distortion and lesions of the bone occurs as a result of hemangiomas, and these bony changes are considered clinically to be very common changes associated with hemangiomas (Waner and Suen, 1999). The bony changes are believed to most likely have been created by the pressure of the hemangioma (Waner and Suen, 1999). A clinical case study involves a 4-day old infant with congenital cranial hemangioma in the right

zygoma, maxilla, frontal, petrous temporal bones, and squamous temporal bone (Koulouris and Rao, 2005). The lesions in this case presented radiologically as both lytic and sclerotic, and were described as having a spoke-wheel appearance (Koulouris and Rao, 2005). Small, osseous hemangioma lesions often are seen radiologically as ill-defined lytic lesions (Koulouris and Rao, 2005).

Ewing's sarcoma is clinically defined as a primary osseous neoplasm that causes skeletal metastases in 10% of patients (Khurana and Fitzpatrick, 2009; Table 8). Ewing's sarcoma is a type of malignant bone tumor that is found frequently in children (Postovsky et al., 2000). Ewing's sarcoma is most commonly found in the long bones of children, and only isolated cases have been reported to be associated with the zygomatic (Postovsky et al., 2000). Ewing's sarcoma has a permeative pattern to the lesion, is formed by primitive mesenchymal round cells, and is mostly found in children and adolescents (Ortner, 2003). Ewing's sarcoma spreads into and through the periosteum, causing the surface bone to respond with reactive bone formation the appearance to be like an onion skin or a radiant sunburst-like form (Ortner, 2003). When viewed radiologically, the lesions are ill defined and lytic with margins that permeate into the surrounding bone (Khurana and Fitzpatrick, 2009).

Another tumor that occurs in children is pilomatricoma (Cecen et al., 2008; Table 8). Pilomatricoma is a benign tumor that can occur at any age, though it is the second most common subcutaneous or cutaneous tumor in children and about 40% of cases occur in the first ten years of age (Cecen et al., 2008). Pilomatricomas are most commonly found in the head and neck in the form of a rock hard mass, though they can occur in other areas such as the zygomatic bones (Cecen et al., 2008). When this type of tumor matures, the cells involved can calcify (Cecen et al., 2008).

Congenital gliomas are benign tumors found in infants (Kern and Macdonald, 1961; Table 8). Congenital gliomas are most commonly found adhering to the nasal processes, but have also been found involving the zygomatic bone (Kern and Macdonald, 1961). The congenital glioma tends to erode the bone it comes into contact with (Kern and Macdonald, 1961). Congenital gliomas have been identified on the malar surface of the zygomatic bone, and have been found to occur unilaterally (Kern and Macdonald, 1961).

Table 11: Bone diseases and neoplasms that occur in the zygomatic bone

Condition	Age	Bilateral Occurrence	Appearance	Location on the Zygomatic Bone	References
Myofibroma	Infant	No	Bone erosion, round and well-defined, sclerotic border	Orbital surface and adjacent external surface	Nam et al., 2005
Intraosseous hemangioma	Infants; including preterm infants	No	Round lesions, destruction of both tables, outward expansion, circular lytic margin, sclerotic, ill-defined	External surface	Moore et al., 2001; Koulouris and Rao, 2005; Ortner, 2003; Waner and Suen, 1999
Ewing's Sarcoma	Children	No	Ill-defined, lytic, reactive bone formation resembles onion skin or sunburst	External surface	Khurana and Fitzpatrick, 2009; Postovsky et al., 2000; Ortner, 2003
Pilomatricoma	Children	Multifocal	Benign, calcification of soft tissue mass	External surface	Cecen et al., 2008
Congenital glioma	Infants	No	Benign tumor, bone erosion	External surface, zygomatic arch	Kern and Macdonald, 1961

Trauma

Trauma to bone can be caused by many factors, and results in fractures due to the abnormal stress placed upon the bone (Ortner, 2003). If the trauma is not fatal, then the

individual will continue to grow, and the bone will undergo modeling and remodeling. Fractures heal more rapidly in children than in adults, but traumatic and pathological fractures vary in the form and time frame in which they heal (Ortner, 2003). Trauma inflicted on the skull will result in different patterns of fractures depending upon different forces involved. There can be bursting fractures that radiate from the compressive site or circular fractures with depressed bone tissue (Ortner, 2003). When fractures occur while the periosteum and other soft tissues are still intact, often small bone fragments will be adherent to the adjacent bone (Ortner, 2003). Considering the fetal and neonatal age range of the sample, the trauma would have had to occur during the birthing process or as a result of the birthing process. In modern clinical practices, intervention during a delivery can include the use of forceps to physically draw the infant out (Ortner, 2003). This can injure the infant, producing a hematoma which may later ossify if the infant survives the birth (Ortner, 2003). Extradural hematoma often occurs in childhood and infancy (Ingram et al., 1949). Extradural hematomas usually occur as a result of some trauma that occurs to the head of the individual, resulting in the lesion and are not always accompanied by a fracture (Ingram et al., 1949).

CHAPTER FIVE: RESULTS

The Temporal Bone Defects

In this sample of twenty-six individuals from the Kellis 2 cemetery, the temporal bone defects were not found in ages younger than about 32 gestational weeks and not older than about 2 months of infancy (Wheeler, 2009; Table 2). In general, the temporal bone defects are more often seen in late term and neonatal individuals in the sample. The abnormality appears to occur bilaterally when both the right and left temporal bones were recovered. The abnormality occurs at the juncture of the petrous and squamous portions on the external surface of the temporal bones around the area of the tympanic or mastoid antrum. The temporal bone abnormality can be described as circular and non-lytic, with an irregular border. There are no fractures associated with the temporal bone abnormalities. The abnormality perforates completely through the temporal bone in some cases, like in the individual from Burial 419 (Figure 22). There is variation to the extent that the abnormality does occur throughout the sample (Figures 16-24). To the lesser extent, the abnormality is smaller in size, and only perforates through the external surface of the bone, and the internal surface of the bone is still intact. Variation can also occur between the squamous and petrous portions of the defect; however, in some individuals the defect has the same appearance in both squamous and petrous portions. The depth of the defect is usually deeper on the petrous portion and shallower on the squamous portion. Variation even occurs between the individual's left and right temporal bones. However, it is unclear whether age has an effect of the appearance of the defect in the temporal bones of the individuals from the Kellis 2 cemetery, considering that the one of the younger individuals, from burial 318b (Figure16), has a more extensive manifestation of the defect than some of the older individuals. There is an even spatial distribution of these

individuals throughout the Kellis 2 cemetery except for the North-Western corner, where there is an apparent absence of individuals with the temporal bone defect (Figure 25).

Figure 16: Left and right temporal bones from the individual from Burial 318b from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. Age at death is estimated to be about 34 weeks gestation. (Image courtesy of S. Wheeler).

Figure 17: The left temporal bone from the individual from Burial 29 from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. Age at death is estimated to be about 38 weeks gestation. (Image courtesy of S. Wheeler).

Figure 18: Left and right temporal bones with defects from the individual from Burial 313 from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. Age at death is estimated to be about 37 weeks gestation (Image courtesy of S. Wheeler)

Figure 19: Left and right temporal bones with defects from the individual from Burial 572 from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. Age at death is estimated to be about 38 weeks gestation. (Image courtesy of S. Wheeler)

Figure 20: The right temporal bone with defect from the individual from Burial 142 from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. Age at death is estimated to be about 39 weeks gestation. (Image courtesy of S. Wheeler)

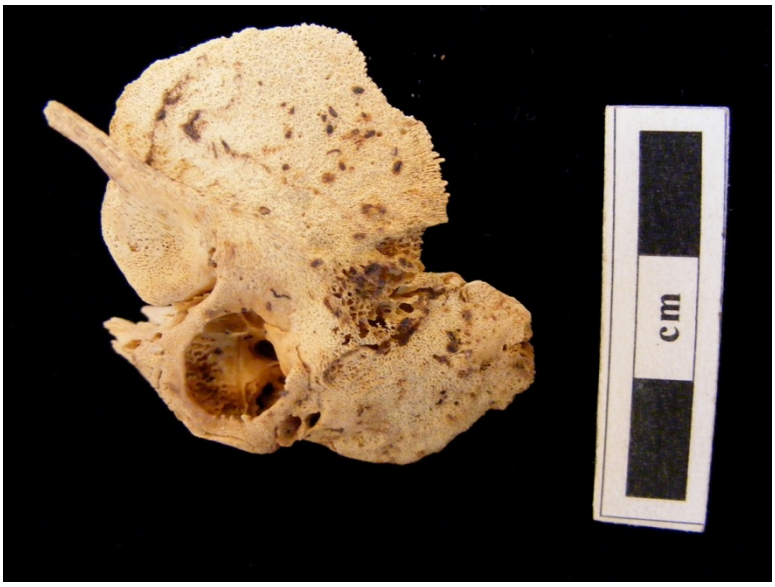


Figure 21: The left temporal bone with defects from the individual from Burial 151 from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. Age at death is estimated to be about 40 weeks gestation. (Image courtesy of S. Wheeler)

Figure 22: Left and right temporal bones with defects from the individual from Burial 419 from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. Age at death is estimated to be about 40 weeks gestation. (Image courtesy of S. Wheeler)

Figure 23: The right temporal bone with defects from the individual from Burial 504 from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. Age at death is estimated to be about 1 month of infancy. (Image courtesy of S. Wheeler)

Figure 24: Left and right temporal bones with defects from the individual from Burial 420 from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. Age at death is estimated to be about 1 month of infancy. (Image courtesy of S. Wheeler)

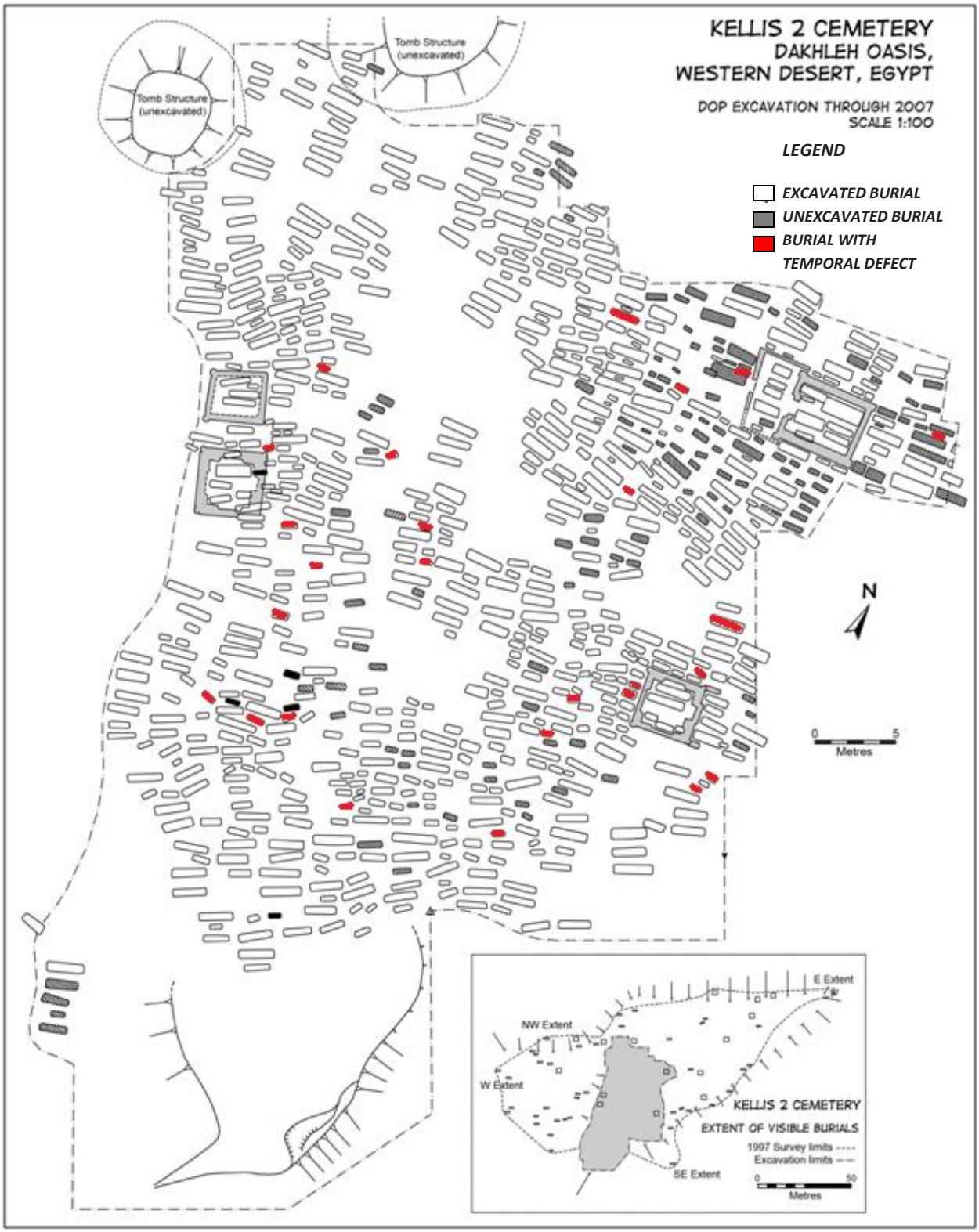


Figure 25: Distribution of the twenty-six individuals of the sample with temporal bone defects within the Kellis 2 cemetery, Dakhleh Oasis, Egypt. (Map adapted from Williams, 2008)

The individuals with the temporal bone defect represent almost 10% of the infant and fetal population recovered from the Kellis 2 cemetery (Table 12). The majority of individuals with the temporal bone defect are considered to be within the fetal age category. Of the 104 fetal individuals recovered from the Kellis 2 cemetery, the individuals with the temporal bone defect represent about 18% of the total fetal population.

Table 12: The distribution of fetal and infant individuals with the temporal bone defects (adapted from Wheeler, 2009).

Age Category	Number of Individuals	Number of Individuals with the Temporal Bone Defect	Percentage
Fetal	104	19	18.3%
Infant	164	7	4.3%
Combined Fetal and Infant	268	26	9.7%

The Zygomatic Bone Lesions

In the sample of six individuals from the Kellis 2 cemetery, the zygomatic bone lesions were not found in ages younger than about 40 gestational weeks (full term). The age of the individuals with the zygomatic bone lesions ranges from about 40 gestational weeks to about 6 months of infancy (Wheeler, 2009; Table 3). The zygomatic bone lesions were therefore found in infants, and not in the younger fetal or gestational age groups (Table 3). The abnormality appears to occur bilaterally when both the right and left zygomatic bones were recovered. The abnormality is located on the external surface of the zygomatic bones, generally from the edge of the orbital surface to or extending past the zygomaticofacial foramen. The zygomatic bone abnormality can be described as a lytic lesion, and the destruction has not perforated completely through the bone but seems localized to the external table. The border of the lesion is irregular. There are no fractures associated with the zygomatic bone abnormalities. There is some evidence of the healing in some of the zygomatic bone lesions as indicated by the

reactive bone formation occurring in some areas of the lesion. Striations can be seen in some of the variations of the zygomatic bone lesion. There is variation in the appearance of the abnormality throughout the sample, from increased porosity to extensive lesions (Figures 26-31). This variation may have a correlation to the age of the individual, for the youngest individual has increased porosity and the older individuals have more developed lesions (Figures 26-31). However, the one of the oldest individuals in the sample that has the zygomatic bone lesions does not have the most extensive or developed version (Figure 30). In fact, the abnormal porosity of the right zygomatic bone of this individual (Burial 390) most clearly resembles closest the abnormal porosity of the youngest individual (Burial 577) (Figure 26). The graves of the six individuals with the zygomatic bone lesions were not evenly distributed throughout the excavated portion of the Kellis 2 cemetery; there is a cluster of three graves in the eastern portion of the Kellis 2 cemetery surrounding a structure (Figure 32). The remaining three are spread through the middle of the cemetery along the west to east axis (Figure 32).

Figure 26: Left and right zygomatic bones of the individual from Burial 577 from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. Age at death is estimated to be about 40 weeks gestation. (Image courtesy of S. Wheeler)

Figure 27: Left and right zygomatic bones of the individual from Burial 580 from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. Age at death is estimated to be about 1 month of infancy. (Image courtesy of S. Wheeler)

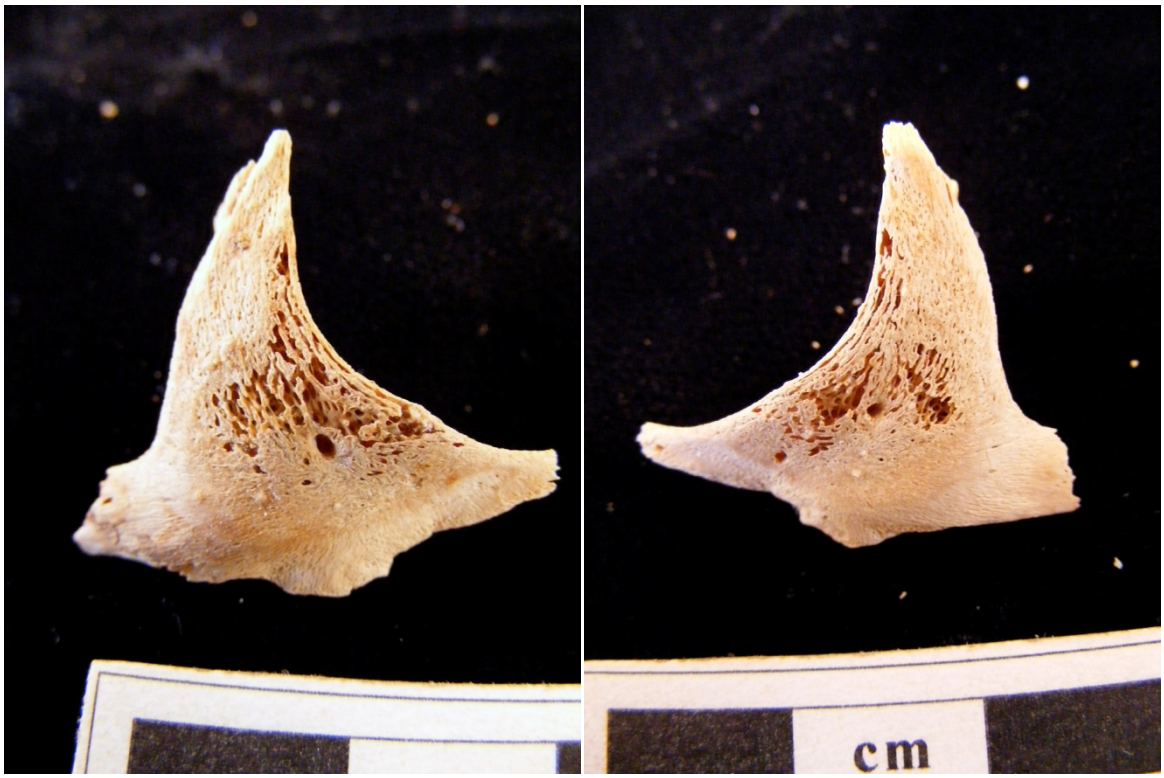


Figure 28: Left and right zygomatic bones of the individual from Burial 118 from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. Age at death is estimated to be about 1 month of infancy. (Image courtesy of S. Wheeler)

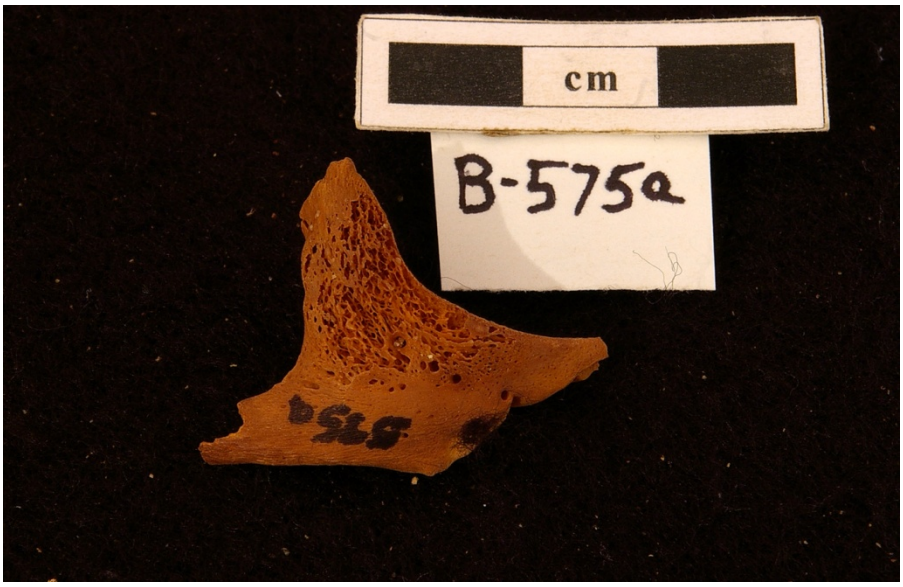


Figure 29: Left zygomatic bone of the individual from Burial 575a from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. Age at death is estimated to be about 2 months old. (Image courtesy of S. Wheeler)

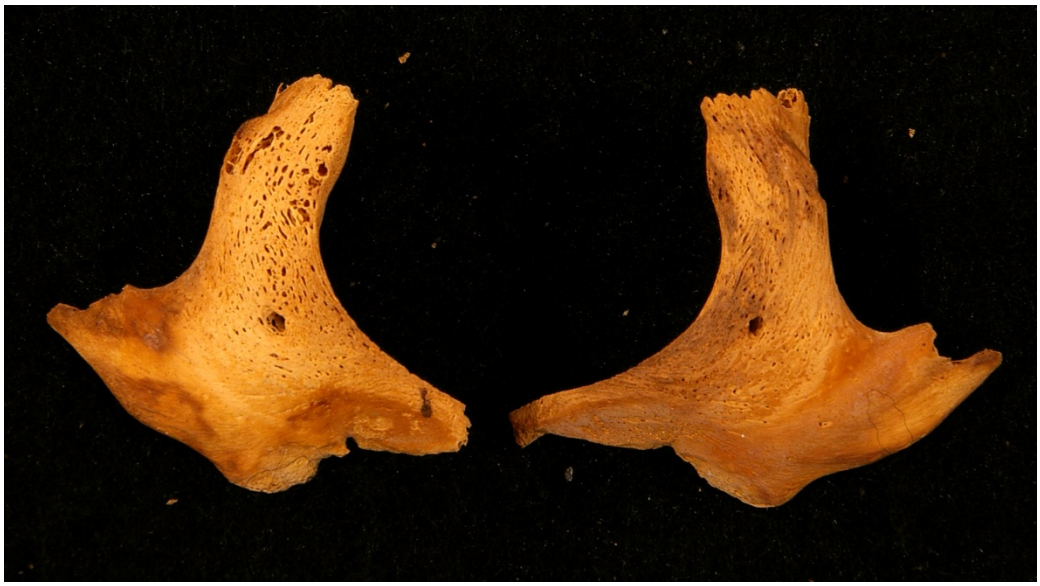


Figure 30: Left and right zygomatic bones of the individual from Burial 390 from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. Age at death is estimated to be about 6 months old. (Image courtesy of S. Wheeler)

Figure 31: Left and right zygomatic bones of the individual from Burial 574 from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. A more extensive version of the zygomatic bone lesion can be seen. Age at death is estimated to be about 6 months old. (Image courtesy of S. Wheeler).

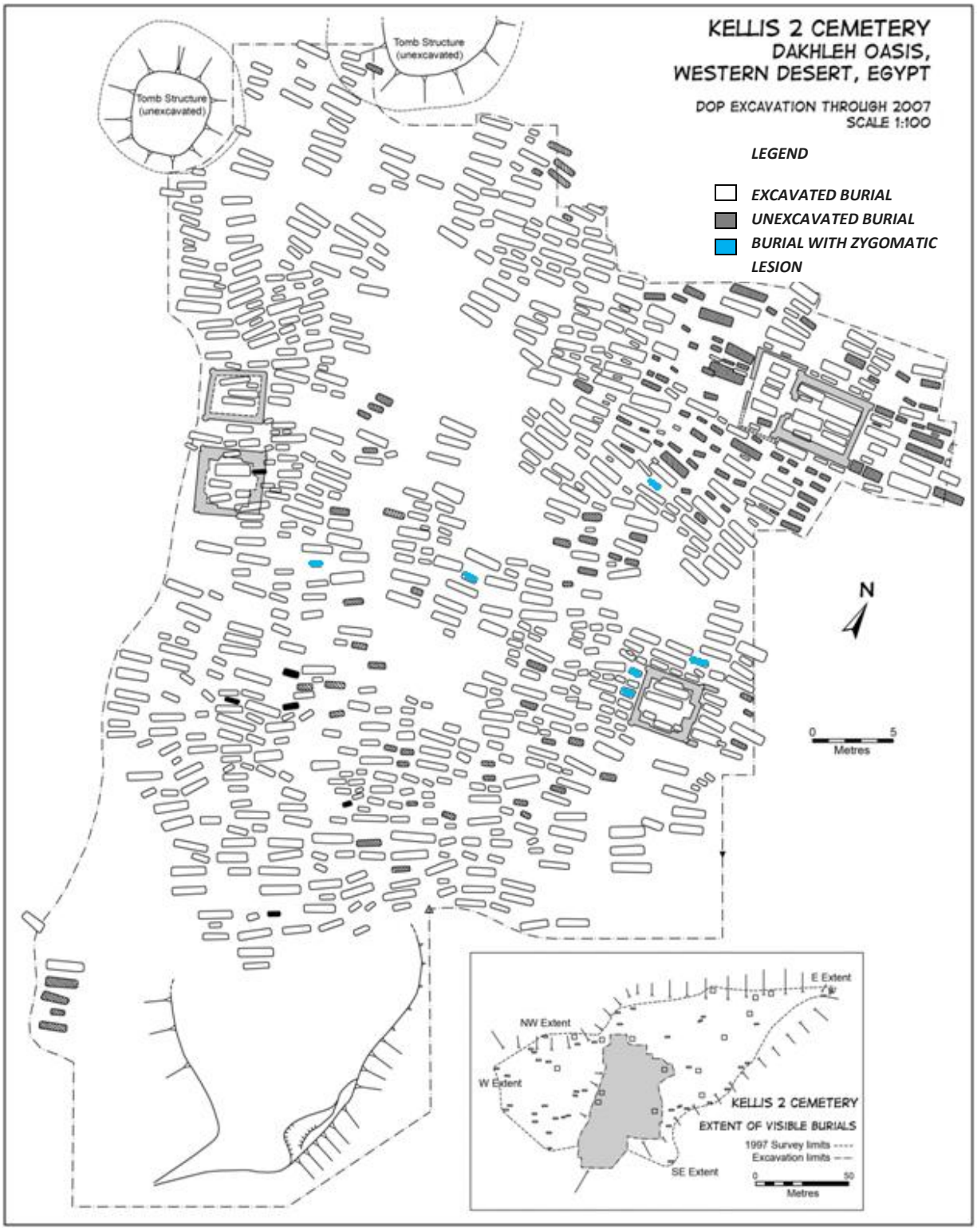


Figure 32: Distribution of the six individuals of the sample with the zygomatic bone lesions within the Kellis 2 cemetery, Dakhleh Oasis, Egypt. (Map adapted from Williams, 2008)

The individuals with the zygomatic bone lesion represent only about 2% of the infant and fetal population recovered from the Kellis 2 cemetery (Table 13). The individuals from the Kellis 2 cemetery that have the zygomatic bone lesion fall solely within the infant category (Table 13); there were about 164 infants recovered from the Kellis 2 cemetery (Wheeler, 2009). Therefore, the individuals with the zygomatic bone lesion represent nearly 4% of the infants recovered from the Kellis 2 cemetery.

Table 13: The distribution of fetal and infant individuals with the zygomatic bone lesions (adapted from Wheeler, 2009).

Age Category	Number of Individuals	Number of Individuals with the Zygomatic Bone Defect	Percentage
Fetal	104	0	0%
Infant	164	6	3.7%
Combined Fetal and Infant	268	6	2.2%

Burial 118 from the Kellis 2 cemetery is the only individual that has both the temporal bone defects and the zygomatic bone lesions. Burial 118 is estimated to be about one month old (Wheeler, 2009), which places the individual within both of the age ranges found for the temporal bone defect group and the zygomatic lesion group. Another unique situation is found in Burial 575, where two individuals were recovered within the same burial. One individual, 575A (estimated age at death of two months old; Wheeler, 2009) has the zygomatic bone lesion and the other, 575B (estimated age at death of 34 weeks gestation; Wheeler, 2009), has the temporal bone defect.

CHAPTER SIX: DIFFERENTIAL DIAGNOSES

In paleopathological analyses, it is important to have both the clinical and anthropological information in order to have all the information needed to make the best-educated differential diagnosis (Aufderheide and Rodriguez-Martin, 1998). The literature survey has provided multiple conditions that can cause abnormalities to occur in the temporal and zygomatic bones of fetal and infant individuals. In order to create a differential diagnosis, these known conditions will be compared to the temporal bone defects and zygomatic bone lesions of the fetal and infant individuals from the Kellis 2 cemetery. The purpose of a differential diagnosis in this paper is to establish which conditions are most likely to have been the cause of the temporal bone defects and zygomatic bone lesions of the individuals from the Kellis 2 cemetery.

Differential Diagnosis of the Temporal Bone Defects

Based upon the appearance, location, and fetal and neonatal occurrence of the temporal bone defects, a differential diagnosis can be established by comparing the information gathered in the literature survey to the macroscopic examination of the individuals from the Kellis 2 cemetery (Table 14). Based upon the age range for this group of individuals from the Kellis 2 cemetery which have the temporal bone defects (Table 2), the only condition that can be excluded based upon age is exostosis; exostosis has not been documented in infant of fetal age individuals (Nemzek and Swartz, 2003). Otitis media and rhabdomyosarcoma are not clinically documented as occurring bilaterally (Fowler, 1940; Freling et al., 2010); the temporal bone defects found on the individuals from the Kellis 2 cemetery are found to occur bilaterally. Many of these conditions occur in both the squamous and petrous portions of the temporal like the defects on the temporal bones of the individuals from the Kellis 2 cemetery. Therefore, the abnormalities caused by these conditions are compared to the temporal bone defects of the

individuals from the Kellis 2 cemetery in order to find similar characteristics. Thus, a differential diagnosis for the temporal bone defects includes the mastoid emissary vein defects and petrosquamous sinus anomalies.

Differential Diagnosis of the Zygomatic Bone Lesions

Based upon the appearance, location, and neonatal and infant occurrence of the zygomatic bone lesion, a differential diagnosis can be established by comparing the information gathered in the literature survey to the macroscopic examination of the individuals from the Kellis 2 cemetery (Table 15). Considering the infant age range of the individuals with the zygomatic bone lesion from the Kellis 2 cemetery (Table 3), all of these conditions have clinical manifestations within the infant age group except for Ewing's sarcoma and pilomatricoma; these two conditions are seen in older children (Ortner, 2003; Cecen et al., 2008). The lesions occur bilaterally in the zygomatic bones of the individuals from the Kellis 2 cemetery; the conditions of myofibroma, intraosseous hemangioma, and congenital glioma have not been documented to occur bilaterally (Nam et al., 2005; Kouliuris and Rao, 2005; Kern and Macdonald, 1961). Considering the many conditions that occur bilaterally and/or multifocally, in order to narrow down the likeliness of the condition to be a possible cause of the zygomatic bone lesions in the sample the characteristics of the abnormalities were compared to those found in the individuals from the Kellis 2 cemetery. This leaves the three pathological conditions of yaws, congenital syphilis, and scurvy. Congenital syphilis and yaws are both treponemal diseases, and therefore the subject of much discussion when it comes to determining their origins throughout the New World, the Old World, and Africa (Rothschild and Heathcote, 1993; Rothschild and Rothschild, 1995; Baker et al., 1988; Crosby, 1969; Mortin and Rashid, 2001; Lobdell and Owsley, 1974; Livingstone, 1991; Barrack, 1956). Geographically, yaws is associated with tropical indigenous populations, and endemic syphilis is associated indigenous populations in drier areas of

subtropical North Africa (Ortner, 2003). Both of these conditions reach the skeleton through the blood stream (Ortner, 2003). However, treponema, in its various forms, is not believed to have been introduced to Egyptian populations earlier than 1400's (Barrack, 1956). Thousands of individuals recovered from ancient Egyptian archaeological sites have been examined and a lack of skeletal evidence of treponemal diseases has been noted (Moller-Christiansen, 1969; Steinbock, 1976). A differential diagnosis of the zygomatic bone lesions of the infant individuals from the Kellis 2 cemetery includes scurvy.

Table 14: Summary of the conditions considered in the differential diagnosis of the temporal bone defects found on the individuals from the Kellis 2 cemetery.

Condition	Age	Bilateral Occurrence	Appearance	Location on the Temporal Bone	References
Mastoiditis	Infants	Yes	Abscesses, one or more walls broken down, increased density	Squamous, petrous portions; mastoid	Graham-Hodgson, 1950; Johnson, 1940
Otitis media	Infants	No	Osteomyelitis, breakdown of cell partitions, sequestrations	Spreads from middle ear to petrous portion	Fowler, 1940; Lindsay, 1945
Streptococcic osteomyelitis	Infants and children	Yes	Acute inflammatory reaction	Spreads from tympanic portion	Ortner, 2003; Boyd-Snee, 1923; Aufderheide and Rodriguez-Martin, 1998
Langerhans' cell histiocytosis	Infants and young children; 1-4 years of age	Yes	Extensive, destructive lytic lesions	Squamous, petrous, and mastoid portions	Fernandez-Latorre et al., 2000; Saliba et al., 2008
Letterer-Siwe disease	Infants; less than 2 years of age	Yes	Osteolytic lesions	Mastoid, petrous, Squamous portions	Balakrishnan et al., 1997; Ortner, 2003
Eosinophilic granuloma	Infants, children and young adults	Yes	Purely lytic, beveled edge, rounded or oval, destructive granuloma lesions of soft tissue and skeleton	Mastoid	Ortner, 2003; Yetiser et al., 2002
Arachnoid granulations	Infants	Yes	Scalloped-edged dural defects, cortical bone erosion	Posterior wall	Lee et al., 2008
Cholesteatoma (acquired and congenital)	Fetal, Infant	Yes	Non-neoplastic destructive lesion, surrounding bone erosion, resorption with slightly sclerotic margins	Anywhere	Persaud et al., 2007; Mays and Holst, 2006; Schmalfuss, 2006
Rhabdomyosarcoma	Fetal, Infant	No	Destructive, osteolytic, metastases, bony changes in surrounding bones as it spreads	First in middle ear, then spreads to mastoid and petrous portions	Freling et al., 2010; Viswanatha, 2007
Exostosis	Children	Yes	Broad based lesions	Along the tympanomastoid, tympanosquamous suture lines	Nemzek and Swartz, 2003; Kern and Macdonald, 1961
Petrosquamous Suture dehiscence	Infant	Yes	Failure of suture to close	Petrous and squamous portion	Boot, 1910; Proctor et al., 1981
Petrosquamous sinus anomalies	Infant	Yes	Enlarged emissary foramen, defect	Petrous portion along the petrosquamous suture	Cheatle, 1900; Chell, 1991; Chauhan et al, 2011; Ruiz et al., 2006
Mastoid emissary vein defect	Infant	Yes	Ovular, enlarged or dilated emissary foramen, defect	Lateral petrous portion, squamous portion	Chauhan et al., 2011

Table 15: Summary of the conditions considered in the differential diagnosis of the zygomatic bone lesions found on the individuals from the Kellis 2 cemetery.

Condition	Age	Bilateral Occurrence	Appearance	Location on the Zygomatic Bone	References
Congenital syphilis	Fetal, Infant, Children	Multifocal	Rounded with destructive foci, hypertrophic periostitis, necrotizing osteitis	External surface	Aufderheide and Rodriguez-Martin, 1998; Ortner, 2003; Khurana and Fitzpatrick, 2009
Tuberculosis	Children	Multifocal	Osteomyelitis	Junction of zygoma and maxilla	Aufderheide and Rodriguez-Martin, 1998; Rothschild and Martin, 2000; Ortner, 2003; Meher et al., 2003
Yaws	Infants, Children	Multifocal	Roughly circular cluster of holes, then central crater-like destruction surrounded by reactive bone formation	External surface	Buckley and Tayles, 2003
Hand-Schuller-Christian disease	Infants, Children	Yes	Defects with destruction of both tables w/o periosteal reaction	Orbital surface	Ortner, 2003; Saliba et al., 2008
Scurvy	Infants, Children	Yes, Multifocal also	Hemorrhagic, inflammatory response, porous lesion, enlargement or thickening of bone tissue; increased porosity; new bone formation	Orbital surface, external and internal surfaces	Roberts and Manchester, 2005; Aufderheide and Rodriquez-Martin, 1998; Brickley and Ives, 2006; Khurana and Fitzpatrick, 2009; Ortner and Ericksen, 1997; Ortner et al., 1999; Mays, 2008; Brown and Ortner, 2011
Myofibroma	Infant	No	Bone erosion, round and well-defined, sclerotic border	Orbital surface and adjacent external surface	Nam et al., 2005
Intraosseous hemangioma	Infants; including preterm infants	No	Round lesions, destruction of both tables, outward expansion, circular lytic margin, sclerotic, ill-defined	External surface	Moore et al., 2001; Koulouris and Rao, 2005; Ortner, 2003; Waner and Suen, 1999
Ewing's Sarcoma	Children	No	Ill-defined, lytic, reactive bone formation resembles onion skin or sunburst	External surface	Khurana and Fitzpatrick, 2009; Postovsky et al., 2000; Ortner, 2003
Pilomatricoma	Children	Multifocal	Benign, calcification of soft tissue mass	External surface	Cecen et al., 2008
Congenital glioma	Infants	No	Benign tumor, bone erosion	Malar surface, zygomatic arch	Kern and Macdonald, 1961

Discussion

The diagnosis of disease in a juvenile population can either be limited or aided due to the preservation, growth and nature of pediatric bone (Lewis. 2007). For example, the skeletal distribution and manifestation of diseases can be different in juveniles than in adults (Lewis, 2007). Incongruities can also occur between what a clinician observes and what a paleopathologist observes due to the fact that a paleopathologist is often dealing with mainly skeletonized materials without soft tissue or family history or the effects of modern medicine (Ortner, 2003). Modern diseases that have not been found yet in the archaeological record should still be considered when performing a differential diagnosis because the lesion cannot be correctly diagnosed if all diseases are not considered (Aufderheide and Rodriguez-Martin, 1998). Different expressions of pathological conditions may also exist between the archaeological record and modern clinical cases due to the effects of modern medicine and technology as well as the evolvement of pathological conditions over time and geographic location (Aufderheide and Rodriguez-Martin, 1998).

When the lesion only affects single bones, it is more difficult to identify the cause of the infection in archaeological cases (Lewis, 2007). When there is a single abnormality, this generally represents a single focus for the pathology in archaeological human skeletal remains, and multiple locations of bony changes indicate a multifocal pathology (Ortner, 2003). The focus of pathology on a single bone means that there is some reason as to why the pathology manifests in that location. The zygomatic bone lesions of the individuals from the Kellis 2 cemetery often engulf the area in which the zygomaticofacial foramen should be seen. If the zygomaticofacial foramen can be seen, the border of the lesion includes the foramen. The bilateral occurrence of the zygomatic bone lesions could therefore reflect the bilateral occurrence of the zygomaticofacial foramen. The effects of scurvy result in abnormal bleeding

or hemorrhaging, and the chronic bleeding leads to an inflammatory response of abnormal and increased vascularity, superficial porosity, and occasionally with hypertrophic bone formation (Ortner, 2003). Postcranial expression of scurvy can also occur in the infantile age group (Ortner and Ericksen, 1997; Ortner, 2003; Brown and Ortner, 2011). Postcranial abnormalities would provide further evidence in support or exclusion of this pathological condition. Subperiosteal hemorrhaging can occur in multiple locations throughout the skeleton, sometimes appearing as subperiosteal hematomas and pathological fractures in the femur and tibia in more extreme cases (Ortner, 2003). The frontal and parietal bones develop enlarged bosses as a reaction to scurvy, and subperiosteal hemorrhaging is commonly found on the frontal bone, especially on its orbital surface (Ortner, 2003). The lacrimal artery crosses the lateral surface of the orbit and also passes through the zygomatic temporal foramen, which if it hemorrhages, could explain the patterning of the lesions on the zygomatic surface without the continuation of the lesions from the orbital surface (Brown and Ortner, 2011). Clinically, retinal hemorrhage is seen in infants with scurvy (Larralde et al., 2007).

Ascorbic acid passes from the mother to the fetus via the placenta unless the mother is suffering from scurvy (Ortner, 2003). Scurvy generally takes a few months before manifesting in a recognizable manner, and is rarely observed before four months of age (Ortner, 2003). Clinical literature is more specific, indicating that scurvy takes one to three months of inadequate vitamin C or ascorbic acid intake to manifest (Larralde et al., 2007).Scurvy in infants younger than three months of age is subject to controversy, but radiological findings have confirmed the subperiosteal hemorrhagic effects of scurvy in infants as young as 5 days old (Hirsch et al., 1976). Scurvy in individuals as young as 5 days old must be the reflection of inadequate maternal supply of ascorbic acid (Hirsch et al., 1976). Due to the latency period of scurvy, a congenital etiology would explain the appearance of scurvy in neonatal infants (Hirsch

et al., 1976). Cooking or boiling milk depletes the ascorbic acid from it (Larralde et al., 2007). Isotopic studies have shown that the infants were consuming animal milk before 6 months of age (Dupras et al., 2001). A diet of animal milk and cereal is a diet that is also low in iron, and has been attributed to anemia and the presence of cribra orbitalia in individuals from the Kellis 2 cemetery (Fairgrieve and Molto, 2000). Individual variation was found to occur in the isotopic analysis of individuals from the Kellis 2 cemetery and was attributed to the fact that the sickly infants would receive a special diet versus healthier infants (Dupras et al., 2001). If the mother was unable to nurse due to death or otherwise, then infants would receive animal milk from birth; this would further exacerbate poor nutrition.

Non-specific indicators of physical stress that manifest on the skull have been subject to much debate as to their etiological origins, whether due to a nutritional deficiency like anemia or scurvy, or other conditions (Ortner, 2003; Ortner, 2001). The lesions of the zygomatic bones can also be considered a non-specific indicator of stress. Abnormal porosity and bone hypertrophy of the orbital surfaces are considered non-specific stress indicators of prolonged periods of stress (Brown and Ortner, 2011). The initial phase of the zygomatic bone lesions appears to be abnormal increased porosity, which is similar to other non-specific indicators of stress such as cribra orbitalia and porotic hyperostosis. Porous and hypertrophic lesions can be caused by many different pathological conditions, but is most commonly linked to anemia (Ortner, 2003). The pathological condition may cause the individual to be anemic, or anemia could simply co-occur with the other pathological condition, since anemia is often connected to the presence of a pathological condition (Ortner, 2003). Non-specific indicators of stress have already been identified in the juvenile and adult individuals from the Kellis 2 cemetery (Cook, 1994; Wheeler, 2009; Wheeler, 2010). The presence of anemia and the connection to cribra orbitalia has also been described in the Kellis 2 cemetery (Fairgrieve and Molto, 2000). In clinical cases, anemia

is the most common hematologic manifestation of scurvy (Tamura et al., 2000). Multiple forms of anemia can occur during pregnancy, including iron-deficiency anemia, and folate deficiency anemia; less commonly are aplastic anemia, hemolytic anemia, thalessemia and sickle cell disease (Sifakis and Pharmakides,2000). Anemia can occur genetically or from iron deficiency. Thalessaemia is the genetic form of anemia; thalessaemia major can result in the appearance of generalized osteopenia throughout the skeleton in an infant, whereas thalessaemia minor has no skeletal manifestations (Lewis, 2010). Thalessaemia affects the frontal bones first, then the facial bones and other cranial bones, and then ribs and postcranial bones (Lewis, 2010). The zygomatic bones are generally described as thickened and bulging in skeletal examples of thalessaemia in infants and children (Lewis, 2010).

The growth and development of the bone affected is important in the consideration of a differential diagnosis. The age group of this sample represents a time when much development and growth is occurring both in the womb and during the first year of life. The temporal bone defects in the individuals from the Kellis 2 cemetery most likely occurred during gestation, and were retained into infancy. Developmental anomalies, variations, or defects can disappear with age, or they can cause malfunctions and resulting health issues. The defect caused by the mastoid emissary vein can result in tinnuitis as well as other complications (Chauhan et al., 2011). Middle ear and other temporal bone infections such as otitis media can spread into the cranial cavity and cause more severe and possibly fatal infections, such as meningitis, when spread through an unclosed petrosquamous suture and through the anomalous route of the petrosquamous sinus (Proctor et al., 1981). The unclosed petrosquamous suture may be a contributing factor in the increased risk of infection and possible mortality, considering the unclosed petrosquamous sutures visible on a majority of the images of the temporal bone defects (Figures 15-23). The petrosquamous suture is normally open at birth (Boot, 1910); the

petrous and squamous portions do not fuse until about one year after birth (Scheuer and Black, 2000). Thus, the petrosquamous suture being open is anatomically normal for this age group of individuals from the Kellis 2 cemetery.

Bone abnormalities can also be representative of normal variation, like the presence of multiple foramina in the zygomatic bone. Bilateral mastoid emissary vein defects and petrosquamous sinus anomalies are residual from embryonic venous tracts (Chauhan et al., 2011). These developmental conditions can represent normal variations of human anatomy. The bone defect created by the mastoid emissary vein can persist into adulthood (Chauhan et al., 2011). If the individual survived infancy and childhood, then in adulthood the residual area from where the emissary vein defect occurred could still be seen. These conditions may also represent a non-metric trait. Familial connections could then be established if it is a non-metric trait.

Sex could also be a factor in the variation in the appearance of the temporal bone defects in the individuals from the Kellis 2 cemetery. Growth varies in individuals due to sex (Scheuer and Black, 2000). Sex can account for different rates of development during gestation; sexual differences can be seen as early as the tenth fetal week (Weaver, 1986). Around the eighth to twelfth week, male fetuses are found to be larger than female fetuses (Pedersen, 1980). The differences in size during gestation are believed to increase as the fetus approaches 40 weeks or full term (Pedersen, 1980).

Infant mortality is generally separated into three categories: late fetal or stillbirth, neonatal, and post-neonatal (Lewis, 2007). Late fetal or still birth and neonatal mortality reflects reflect the endogenous state of the infant as affected by the maternal and genetic influences, and post-neonatal mortality reflects the exogenous state of the infant as affected by the external

environment's influences (Lewis, 2007). This would suggest the possibility that if the individuals are from the fetal age group that they could represent endogenous mortality instead of exogenous mortality. The estimated age at death for the individuals with the temporal bone defects ranges from the fetal period to infancy (Table 10, Figure 13). The majority of individuals (20 out of 26) have an estimated age at death that is 40 gestational weeks and younger (Table 10; Figure 13); considering the premature deaths of these individuals the temporal bone defect developed during gestation since there would have been no feasible time for it to develop after delivery, especially so in the case of preterm delivery. The temporal bone defect may have increased the risk of mortality in the first month of life if infant is exposed to infection, and then the temporal bone defects may have closed and healed with age or resulted in infant mortality; two months of age was the oldest estimated age at death for the individuals with the temporal bone defects (Table 10, Figure 13). Thus, the temporal bone defect is likely to be a result of endogenous factors leading to the death of these individuals. A developmental condition, such as petrosquamous sinus anomalies and mastoid emissary vein defects, would occur during gestational development and would also agree with the likelihood that endogenous factors caused the temporal bone defects in these individuals from the Kellis 2 cemetery.

The estimated age at death for the individuals with the zygomatic bone lesions ranges from 40 weeks gestation to 6 months of infancy (Table 11, Figure 14). Births resulting in viable individuals are likely after 38 weeks gestation; whether or not the youngest individual with the zygomatic bone lesions was born prematurely and then lived two more weeks before death is undeterminable. This age range indicates that the lesions are more likely attributed to exogenous factors that the infant would be exposed to after birth, though the youngest individual indicates that the endogenous factors of the individuals must not be eliminated as a cause for the zygomatic bone lesions. A nutritional deficiency can begin in the womb due to the nutritional

deficiency of the mother, and then can be further exacerbated by continued nutritional deficiency after birth. The differential diagnosis of scurvy is concurrent with the likelihood that both endogenous and exogenous factors contributed to the occurrence of the zygomatic bone lesions of the individuals from the Kellis 2 cemetery.

The individuals in this study represent those individuals who had the temporal bone defects and zygomatic bone lesions in the given age groups that have died. It is unknown if other individuals had the same temporal bone defects and zygomatic bone lesions and survived into adulthood. Perhaps with adulthood, these abnormalities would no longer be visible. Therefore, since the whole population is not represented in just the deceased individuals, the estimations of prevalence of these pathological conditions within the population are subject to a selectivity bias (Wood et al., 1992). This is because individuals have different experiences of health and illness, and therefore the deceased population cannot represent the living population in the prevalence of pathological conditions (Wright and Yoder, 2003).This also means that there is a hidden heterogeneity in risks within the population, since individuals have varying levels of susceptibility to disease and death (Wood et al., 1992). All the factors that cause an individual's susceptibility to disease and death generally cannot be identifiable (Wright and Yoder, 2003). If more research is conducted on the individuals recovered from the Kellis 2 cemetery, then more factors that contribute to the individual's susceptibility to pathological conditions can be identified.

CHAPTER SEVEN: CONCLUSION AND FUTURE CONSIDERATIONS

Summation

The excavation of the individuals from the Kellis 2 cemetery has so far resulted in around 268 individuals aged less than one year (Wheeler, 2009). The individuals recovered from the Kellis 2 cemetery included infant and fetal individuals who had undiagnosed abnormalities on their zygomatic and temporal bones. In order to create a differential diagnosis for both abnormalities, a literature survey was conducted in order to collect information on the known causes of abnormalities on the zygomatic and temporal bones in the infant and fetal age group. The growth and development of the zygomatic and temporal bones were also taken into consideration for possible causation. The differential diagnosis of the defects on the temporal bones of the twenty-six individuals from the Kellis 2 cemetery includes the mastoid emissary vein defect and anomalies of the petrosquamous sinus. The differential diagnosis was based on the known causes of abnormalities that occur in the temporal bones of fetal and infant individuals in clinical and paleopathological literature, as well as a comparison of the appearance, location, and bilateral occurrence of the abnormality in the temporal bone. The fact that the petrosquamousal suture is unclosed at birth may have increased the potential for mortality. The differential diagnosis of the lesions on the zygomatic bones of the six individuals from the Kellis 2 cemetery includes scurvy. The differential diagnosis was based on the known causes of abnormalities that occur in the zygomatic bones of infant individuals in clinical and paleopathogical literature, as well as a comparison of the appearance, location, and bilateral occurrence of the abnormality in the zygomatic bone. Pathological conditions were also excluded based upon geographical and historical likelihood of occurrence in Northern Africa and Egypt. Non-specific indicators of physical stress can also be attributed to the effects of scurvy and the anemia that tends to occur in individuals with scurvy.

These two unrelated conditions seen in the individuals from the Kellis 2 cemetery have previously not been reported in juvenile paleopathological literature. The differential diagnoses of the temporal bone defects and zygomatic bone lesions provide additional information to the growing literature on paleopathology in juvenile individuals. The most direct evidence about juvenile individuals is gathered from the examination of their physical skeletal remains (Lewis, 2007). With each paleopathological condition that can be identified, more information can be learned about the past lives of the juvenile individuals.

Future Considerations

Macroscopic observation was the sole research method for this study, but in the future more intensive methods of study can be employed in order to provide additional information on the individuals who have the temporal bone defects and zygomatic bone lesions. Future research could involve radiographs or CT imaging, microscopic study, chemical analysis, and genetic testing. By gaining more information on the individuals who have the temporal bone defects and zygomatic bone lesions, a clearer picture can be presented on the health status of those individuals as well as the nature of the condition. The evidence of disease is part of a larger assessment of the health status on juveniles that combines with nutritional stress and growth data in order to provide information on the ability to adapt to certain ecological or cultural conditions (Lewis, 2007).

Radiography or CT imaging (computed tomography) is a non-destructive method of observation that allows the internal effects of the pathological condition to be visualized. The pattern of bone density within the lesion or defect and the area around it can be visualized with the use of radiography or CT imaging. The density of the bones affected by the abnormalities can have implications in creating a differential diagnosis. The bone density could be due to the

effects of nutrition and stress as well as the effects of the lesion or defect. For example, bony demineralization is the most common manifestation of scurvy that is seen in radiographs (Tamura et al., 2000).

Microscopic study is a destructive method of observation that also allows an internal view at the effects of the bone lesion or defect. Microscopic study could potentially help determine if there is any decreased, increased, or normal osteoblastic activity that exists in combination with any decreased, increased, or normal osteoclastic activity. The abnormal variation that is found can be indicative of certain pathological conditions.

A chemical analysis of the bones involved could determine the existence of nutritional deficiencies. A vitamin or nutritional deficiency could be the cause of the temporal bone defect or the zygomatic bone lesions; or at least the cause of the susceptibility to these abnormalities. The individual receives vitamins and nutrients during gestation from the mother, and these essential elements are stored in order to aid the individual in the first few months of life (Lewis, 2007). There are individuals that are considered to be premature in the sample of individuals with the temporal bone defects. Prematurity limits the amount of essential vitamins and nutrients the infant can store since the gestational period is shorter than the average forty weeks (Lewis, 2007). Therefore, a chance exists that these premature individuals in this sample may have had deficiency diseases. The older individuals could have also been premature but survived longer, and could also have had a predisposition to developing deficiency diseases.

If genetic testing was performed on individuals of the sample, the results could have an impact on the differential diagnosis if a familial connection could be established or if a specific gene could be identified. A genetic condition could therefore be the more likely cause instead of other non-genetic conditions. If the mastoid emissary vein defects and petrosquamous sinus

anomalies are non-metric traits, then a familial connection could be made among the twenty-six individuals from the Kellis 2 cemetery that have the temporal bone defects. There is congenital condition that has similarities to the temporal bone defects in this sample, though the condition occurs in different bones of the skull. Enlarged parietal foramina are slightly larger than normal parietal foramina, and also occur bilaterally just like the smaller version, and are considered a normal variant in the ossification process to allow the passage of emissary veins (Reddy et al., 2000).Congenital enlarged parietal foramina are found to commonly occur bilaterally, located near the sagittal suture, and are considered anomalies that most likely result from autosomal dominant genes as well as a possible familial relationship (Aufderheide and Rodriguez, 1998; Ortner, 2003). The enlarged parietal foramina are considered to be a benign anomaly (Reddy et al., 2000). The diameter of the enlarged foramina surpasses the average size of 1-2 mm (Aufderheide and Rodriguez, 1998). The perforations are located on the posterior portion of both parietals near the sagittal suture (Ortner, 2003). This condition occurs near the sutures and bilaterally (Ortner, 2003).

Future research should also involve the macroscopic observation of other infant and fetal populations whose skeletal remains have been recovered from other archaeological sites. Previous research may have overlooked these temporal bone defects and zygomatic bone abnormalities. By occurring in different populations, trends could then be established based upon the time period, demographics, climate, nutrition availability, and geographic location. This would also have implications in creating a differential diagnosis.

One individual with the temporal bone defect was found in a sample of fetuses from some Nubian pot burials from Askut, Sudan. Nubia is believed to have been the area from Wadi Halfa in the north to Khartoum in the south, and the Nubian fetal pot burials are from an area

south of Aswan near the first and second cataracts (Britton, 2009). The archaeological site of Askut is believed to have been occupied during the New Kingdom and Third Intermediate period, around 1850 BC – 1070 BC (Britton, 2009). Nubia was an Egyptian colony during this time period, and Nubian culture was affected by Egyptian ideologies and burial practices (Britton, 2009). The individual from burial 2186 from the Nubian fetal pot burial collection appears to also have the temporal bone defect, though it appears unilaterally and not in as an advanced state as the individuals from the Kellis 2 cemetery, Dakhleh Oasis, Egypt. This individual has been estimated to be between 36-40 gestational weeks (Britton, 2009). The late gestational age range is similar to the age range of those individuals with the temporal bone defect from the Kellis 2 cemetery.

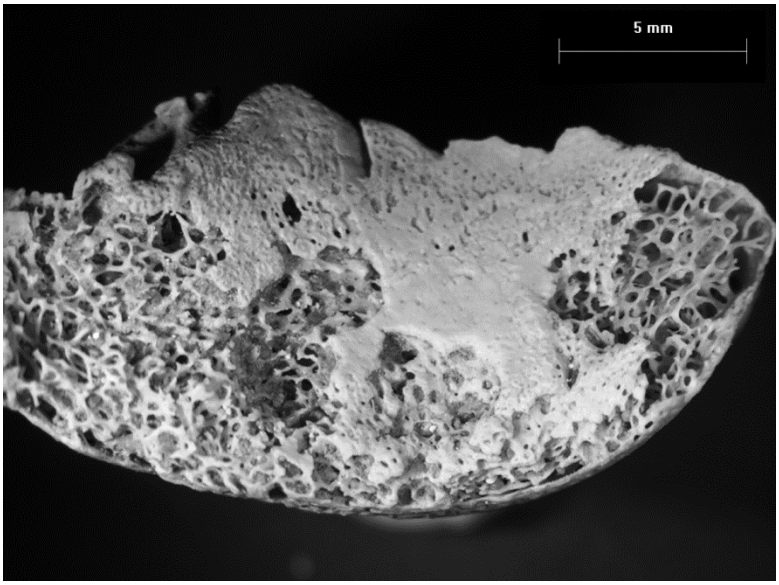
APPENDIX A:
ADDITIONAL IMAGES OF THE TEMPORAL BONE DEFECTS

Burial 701 (Image courtesy of S. Wheeler)

Burial 518 (Image courtesy of S. Wheeler)

APPENDIX B:
ADDITIONAL IMAGES OF THE ZYGOMATIC BONE LESIONS

Orbital surface of zygomatic bone from individual 575a (Image courtesy of S. Wheeler)



Orbital surface of the right zygomatic bone from individual 580 (Image courtesy of S. Wheeler)

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